



This is a digital copy of a book that was preserved for generations on library shelves before it was carefully scanned by Google as part of a project to make the world's books discoverable online.

It has survived long enough for the copyright to expire and the book to enter the public domain. A public domain book is one that was never subject to copyright or whose legal copyright term has expired. Whether a book is in the public domain may vary country to country. Public domain books are our gateways to the past, representing a wealth of history, culture and knowledge that's often difficult to discover.

Marks, notations and other marginalia present in the original volume will appear in this file - a reminder of this book's long journey from the publisher to a library and finally to you.

Usage guidelines

Google is proud to partner with libraries to digitize public domain materials and make them widely accessible. Public domain books belong to the public and we are merely their custodians. Nevertheless, this work is expensive, so in order to keep providing this resource, we have taken steps to prevent abuse by commercial parties, including placing technical restrictions on automated querying.

We also ask that you:

- + *Make non-commercial use of the files* We designed Google Book Search for use by individuals, and we request that you use these files for personal, non-commercial purposes.
- + *Refrain from automated querying* Do not send automated queries of any sort to Google's system: If you are conducting research on machine translation, optical character recognition or other areas where access to a large amount of text is helpful, please contact us. We encourage the use of public domain materials for these purposes and may be able to help.
- + *Maintain attribution* The Google "watermark" you see on each file is essential for informing people about this project and helping them find additional materials through Google Book Search. Please do not remove it.
- + *Keep it legal* Whatever your use, remember that you are responsible for ensuring that what you are doing is legal. Do not assume that just because we believe a book is in the public domain for users in the United States, that the work is also in the public domain for users in other countries. Whether a book is still in copyright varies from country to country, and we can't offer guidance on whether any specific use of any specific book is allowed. Please do not assume that a book's appearance in Google Book Search means it can be used in any manner anywhere in the world. Copyright infringement liability can be quite severe.

About Google Book Search

Google's mission is to organize the world's information and to make it universally accessible and useful. Google Book Search helps readers discover the world's books while helping authors and publishers reach new audiences. You can search through the full text of this book on the web at <http://books.google.com/>



International clinics

Medical Library

BOSTON
MEDICAL LIBRARY
8 THE FENWAY

The Gift of

Date



INTERNATIONAL CLINICS

A QUARTERLY

OF

ILLUSTRATED CLINICAL LECTURES AND
ESPECIALLY PREPARED ARTICLES

ON

MEDICINE, NEUROLOGY, SURGERY, THERAPEUTICS, OB-
STETRICS, PÆDIATRICS, PATHOLOGY, DERMATOLOGY,
DISEASES OF THE EYE, EAR, NOSE, AND THROAT,
AND OTHER TOPICS OF INTEREST TO
STUDENTS AND PRACTITIONERS

BY LEADING MEMBERS OF THE MEDICAL PROFESSION
THROUGHOUT THE WORLD

EDITED BY

HENRY W. CATTELL., A.M., M.D., PHILADELPHIA, U.S.A.

WITH THE COLLABORATION OF

JOHN B. MURPHY, M.D., ALEXANDER D. BLACKADER, M.D.,
CHICAGO MONTREAL

H. C. WOOD, M.D., T. M. ROTCH, M.D., E. LANDOLT, M.D.,
PHILADELPHIA BOSTON PARIS

THOMAS G. MORTON, M.D., JAMES J. WALSH, M.D.,
PHILADELPHIA NEW YORK

J. W. BALLANTYNE, M.D., AND JOHN HAROLD, M.D.,
EDINBURGH LONDON

WITH REGULAR CORRESPONDENTS IN MONTREAL, LONDON,
PARIS, LEIPSIK, AND VIENNA

41.A218

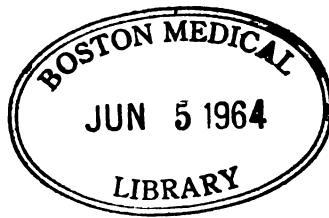
VOLUME IV. TWELFTH SERIES, 1903

PHILADELPHIA

J. B. LIPPINCOTT COMPANY

1903

COPYRIGHT, 1903
BY
J. B. LIPPINCOTT COMPANY



PRINTED BY J. B. LIPPINCOTT COMPANY, PHILADELPHIA, U.S.A.

CONTRIBUTORS TO VOLUME IV.

(TWELFTH SERIES.)

BISHOP, E. STANMORE, F.R.C.S. (Eng.), Honorary Surgeon, Ancoats Hospital, Manchester; President of the Manchester Clinical Society; Fellow of the British Gynæcological Society, etc.

BODINE, J. A., M.D., Adjunct Professor of Surgery at the New York Polyclinic; Attending Surgeon at St. John's Hospital, Long Island City, New York, etc.

BOSANQUET, WILLIAM CECIL, M.A., M.D. (Oxon.), M.R.C.P. (Lond.), Physician to the Out-Patient Department at the Victoria Hospital for Children, Chelsea; Pathologist to Charing Cross Hospital.

BROWN, THOMAS R., M.D., Instructor in Medicine, Johns Hopkins Medical School, Baltimore, Md.

CHASE, ROBERT H., A.M., M.D., Superintendent of Friends Asylum, Frankford, Philadelphia.

EISENDRATH, DANIEL N., M.D., Professor of Clinical Anatomy, College of Physicians and Surgeons (Medical Department of University of Illinois); Professor of Surgery, Post-Graduate Medical School; Attending Pathologist, Michael Reese Hospital, etc., Chicago.

FALLON, M. F., M.D., Attending Surgeon to St. Vincent's Hospital, Worcester, Mass.

FLICK, LAWRENCE F., M.D., Philadelphia.

GARDINER, CHARLES FOX, M.D., Colorado Springs, Colorado.

HALL, J. N., M.D., Professor of Medicine in the Denver and Gross College of Medicine.

HENRY, FREDERICK P., A.M., M.D., Professor of the Principles and Practice of Medicine, and of Clinical Medicine, in the Woman's Medical College of Pennsylvania; Physician to the Philadelphia Hospital.

HINSDALE, GUY, A.M., M.D., President of the Pennsylvania Society for the Prevention of Tuberculosis.

KELLY, ALOYSIUS O. J., A.M., M.D., Instructor in Clinical Medicine and Assistant Physician to the Hospital, University of Pennsylvania; Professor of the Theory and Practice of Medicine, University of Vermont; Physician to St. Mary's and St. Agnes's Hospitals, and Pathologist to the German Hospital, etc., Philadelphia.

LANCEREAUX, E., M.D., Physician to the Paris Hospitals.

LESER, EDMUND, M.D., Professor of Surgery at the University of Halle.

MOYNIHAN, BERKELEY G. A., M.S. (Lond.), F.R.C.S. (Eng.), Assistant Surgeon, Leeds General Infirmary.

PACKARD, THE LATE FREDERICK A., A.M., M.D., Philadelphia.

SANTI, PHILIP R. W. DE, F.R.C.S., Surgeon Laryngologist and Aural Surgeon to Westminster Hospital; Late Senior Assistant Surgeon to Westminster Hospital, London.

SCHLAPP, MAX, M.D., Lecturer on Nervous Pathology at Cornell Medical School; Attendant Neurologist to the Dispensary of the Presbyterian Hospital, etc., New York.

SHOEMAKER, WILLIAM T., M.D., Assistant Ophthalmologist and a Chief of Clinics to the German Hospital; Dispensary Ophthalmic Surgeon to the Presbyterian Hospital, Philadelphia.

SPILLER, WILLIAM G., M.D., Assistant Professor of Nervous Diseases and Assistant Professor of Neuropathology in the University of Pennsylvania; Neurologist to the Philadelphia Hospital.

STERN, HEINRICH, PH.D., M.D., Professor of Internal Medicine at the New York School of Clinical Medicine; Visiting Physician to the Red Cross, St. Elizabeth's, and Metropolitan Hospitals of New York City.

STEWART, PURVES, M.D., M.R.C.P., Assistant Physician to Westminster Hospital, London.

VAN HARLINGEN, ARTHUR, M.D., Philadelphia.

CONTENTS OF VOLUME IV.

(TWELFTH SERIES.)

THERAPEUTICS.

	PAGE
THE SANATORY TENT AND ITS USE IN THE TREATMENT OF PULMONARY TUBERCULOSIS. By CHARLES FOX GARDINER, M.D.....	1
THE TREATMENT OF CHRONIC GASTRIC CATARRH. By HEINRICH STERN, PH.D., M.D.....	7
SOME PRACTICAL POINTS ON THE EARLY DIAGNOSIS AND TREATMENT OF MALIGNANT DISEASE OF THE LARYNX. By PHILIP R. W. DE SANTI, F.R.C.S.....	28
TREATMENT OF ANEURISMS BY GELATIN IN HYPODERMIC INJECTIONS. By E. LANCEREAUX, M.D.....	36
SELECTED PRESCRIPTIONS	39

MEDICINE.

REPORT OF A CASE OF PERNICIOUS ANÆMIA WITH EXTENSIVE PIGMENTARY CHANGES IN THE SKIN. By THE LATE FREDERICK A. PACKARD, A.M., M.D.....	47
EXHIBITION OF LUNGS AND HEART FROM A CASE OF LEFT-SIDED PNEUMONIA WITH PURULENT PERICARDIAL EFFUSION. By FREDERICK P. HENRY, A.M., M.D.....	51
DIFFERENTIAL DIAGNOSIS BETWEEN TUBERCULOSIS OF THE LUNGS AND DISEASES WHICH RESEMBLE IT. By LAWRENCE F. FLICK, M.D.....	56
ABDOMINAL DIAGNOSIS. By E. STANMORE BISHOP, F.R.C.S.....	63
SCARS AND MARKS OF CLINICAL INTEREST. By J. N. HALL, M.D..	71
THE NATURE OF CANCER AND OTHER NEW GROWTHS. By WILLIAM CECIL BOSANQUET, M.A., M.D. (Oxon.), M.R.C.P. (Lond.).....	77
SOME CLINICAL ASPECTS OF ANEURISMS OF THE AORTA. By ALOYSIUS O. J. KELLY, A.M., M.D.....	81

NEUROLOGY.

SOME ASPECTS OF PARANOIA. By ROBERT H. CHASE, A.M., M.D.....	97
TRAUMATIC LESIONS OF THE BRAIN IN THEIR RELATION TO OPERATION. By WILLIAM G. SPILLER, M.D.....	102

	PAGE
THE DIAGNOSIS OF FUNCTIONAL AND ORGANIC HEMIPLEGIA. By PURVES STEWART, M.D., M.R.C.P.....	112
SYPHILITIC HEMIPLEGIA; LEAD PALSY AND TRAUMA; ANOM- ALOUS GENERAL PARALYSIS; HYSTERICAL MUTISM. By MAX SCHLAPP, M.D.....	122

SURGERY.

ANATOMY OF THE INGUINAL REGION AND THE RADICAL CURE OF INGUINAL HERNIA. By M. F. FALLON, M.D.....	136
THE SURGICAL TREATMENT OF HÆMATEMESIS FROM GASTRIC ULCER. By BERKELEY G. A. MOYNIHAN, M.S. (Lond.), F.R.C.S. (Eng.)	144
FRIGHT AND DEATHS IN CHLOROFORM NARCOSIS; MENTAL PREOCCUPATION AS A PRELIMINARY TO GENERAL ANÆS- THESIA; RESECTION OF TUBERCULOUS TESTICLE. By J. A. BODINE, M.D.	153
A CASE OF MELOPLASTY; TUBERCULOUS GLANDS OF THE NECK. By DANIEL N. EISENDRATH, M.D.....	164
ARTIFICIAL RESPIRATION AND HEART MASSAGE IN NARCOSIS; VOLKMANN'S SUSPENSION APPARATUS; LIGATURE OF THE COMMON CAROTID ARTERY; OPENING OF THE FRONTAL SINUS; RESECTION OF THE SUPERIOR AND INFERIOR ORBI- TAL NERVES; SUTURING OF THE BOWEL. By EDMUND LESER, M.D.	174

DERMATOLOGY.

RECURRING PHLYCTENULAR ERUPTION OF THE FINGERS, WITH CHANGES IN THE NAILS, POSSIBLY OF HYSTERICAL ORIGIN. By ARTHUR VAN HARLINGEN, M.D.....	180
---	-----

OPHTHALMOLOGY.

THE CLINICAL SIGNIFICANCE OF BINOCULAR DIPLOPIA. By WILLIAM T. SHOEMAKER, M.D.....	188
---	-----

BIOGRAPHICAL SKETCHES OF EMINENT LIVING PHYSICIANS.

HORATIO C. WOOD, M.D., LL.D., AND WILLIAM W. KEEN, M.D., LL.D. By GUY HINSDALE, A.M., M.D.	198
--	-----

MONOGRAPH.

THE BLOOD IN HEALTH AND IN DISEASE, WITH A REVIEW OF THE RECENT IMPORTANT WORK ON THIS SUBJECT. By THOMAS R. BROWN, M.D.....	205
--	-----

LIST OF ILLUSTRATIONS TO VOLUME IV.

(TWELFTH SERIES.)

PLATES.

	PAGE
The Gardiner Sanatory Tent (Plates Nos. 1, 2, 3, and 4)	2-3
Exterior of a Dr. Gardiner's Sanatory Tent situated in the pines (Fig. 5)	4
Interior of tent furnished as an ordinary room (Fig. 6)	5
Dr. Stern's apparatus for irrigation of the stomach (Fig. 1)	18
Method of using Dr. Stern's apparatus in irrigation of the stomach (Fig. 2)	19
Pernicious anæmia with marked pigmentary changes in the skin, posterior view (Fig. 1)	48
Anterolateral view of the same patient (Fig. 2)	49
Left-sided pneumonia with purulent pericardial effusion (Fig. 1)	55
Posterior view. Maximum points; Areas (Fig. 1); Lateral maximum points (Fig. 2)	66
Anterior view. Maximum points; Areas (Fig. 3); Lateral areas (Fig. 4)	68
Connection between sympathetic nerves supplying viscera and spinal nerves supplying muscles of abdominal walls (Fig. 5)	70
Large aneurism of the transverse portion of the arch of the aorta (Fig. 1); Large aneurism of the arch of the aorta with erosion of the sternum (Fig. 2); Double aneurism of the arch of the aorta, with sudden death from rupture into the œsophagus (Fig. 3)	84
Aneurism of the arch of the aorta protruding through the sternum (Fig. 4); Aneurism of the arch of the aorta protruding through the sternum (Fig. 5)	85
Cast made after death from external rupture of the aneurism illustrated in Figs. 4, 5, and 7 (Fig. 6)	86
Large aneurism of the arch of the aorta protruding through and causing almost complete absorption of the sternum and adjacent costal cartilages (Fig. 7)	87
Large dissecting aneurism of the abdominal aorta (Fig. 8)	88
Classical paranoia (Fig. 1); Persecutory type of paranoia (Fig. 2); Homicidal type of paranoia (Fig. 3); Erotic type of paranoia (Fig. 4)	98
Jealous type of paranoia (Fig. 5); Querulous type of paranoia (Fig. 6); Ambitious type of paranoia (Fig. 7); Religious type of paranoia (Fig. 8); Primary paranoia (Fig. 9); Hebephrenia paranoides (Fig. 10); Rudimentary type (Fig. 11); Reasoning paranoia (Fig. 12) ...	100

	PAGE
Old infantile hemiplegia (Fig. 1); Hysterical hemiplegia; showing glosso-labiobrachial spasm of left side (Fig. 4).....	116
Relation of blood-vessels and nerves to line of incision in the radical cure of inguinal hernia (Fig. 1).....	140
Right inguinal canal under normal conditions, showing muscular and fascial relations (Fig. 2).....	142
Floor and neighboring deep structures of inguinal canal, showing tissues that come into view in the operation for the radical cure of inguinal hernia (Fig. 3).....	143
Appearance of patient previous to a plastic operation for deformity resulting from the removal of a tumor of the cheek.....	164
Appearance of patient after the operation.....	165
Method of performing artificial respiration (Figs. 1 and 2).....	174
Superior tracheotomy (Fig. 3); Volkmann's suspension apparatus (Fig. 4).....	175
Ligature of the common carotid (Fig. 5); Ligature of the posterior tibial artery (Fig. 6).....	176
Opening of the frontal sinus; Resection of the supra-orbital and infra-orbital nerves (Fig. 7); Resection of the bowel (Figs. 8, 9, and 10)	177, 178
Sutures used in Bassini's radical operation for inguinal hernia (Fig. 11) ..	179
Recurring phlyctenular eruption of fingers (Figs. 1 and 2).....	180
Compensatory position of the head when the field of diplopia is to the left (Fig. 5); When the field of diplopia is up and to the left (Fig. 6); When the field of diplopia is down and to the right (Fig. 7).....	196
Horatio C. Wood, M.D., LL.D. (Fig. 1).....	198
William W. Keen, M.D., LL.D. (Fig. 1).....	202
Dr. William W. Keen operating at the Jefferson Medical College (Fig. 2) ..	203

FIGURES.

Faucet in Dr. Stern's apparatus for irrigation of the stomach (Fig. 3)...	19
Area of dulness in aneurism of the descending portion of the arch of the aorta (Fig. 9); Of the ascending portion of the arch of the aorta (Fig. 10)	89
Crossed amblyopia (Fig. 2).....	118
Hysterical hemiplegia; areas of anæsthesia on paralyzed side (Fig. 3)...	119
The eye fixing the object <i>A</i> (Fig. 1); the two eyes fixing on <i>A</i> (Fig. 2) ..	193
The right eye fixing <i>A</i> and the left eye turned in so that its visual axis takes the direction <i>FC</i> (Fig. 3).....	194
The right eye fixing <i>A</i> , the left eye divergent (Fig. 4).....	195

Therapeutics

THE SANATORY TENT AND ITS USE IN THE TREATMENT OF PULMONARY TUBERCULOSIS.

BY CHARLES FOX GARDINER, M.D.,

Of Colorado Springs, Colorado.

THE cure of consumption, to-day, rests absolutely upon one great factor in treatment, and that is—out-door air. Any method by which a consumptive can breathe out-door air, day and night, without exposure and without fatigue, and surrounded by the comforts or even luxuries so necessary to the invalid's existence, should be warmly embraced by all who are interested in the cure of this terrible disease.

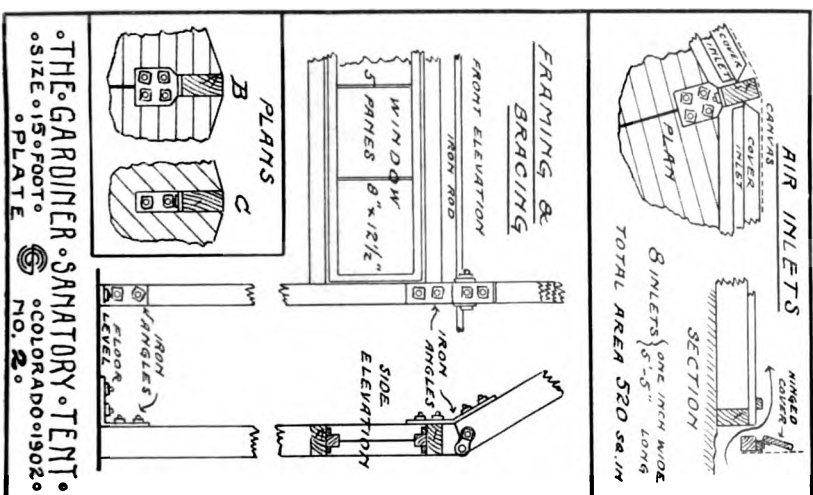
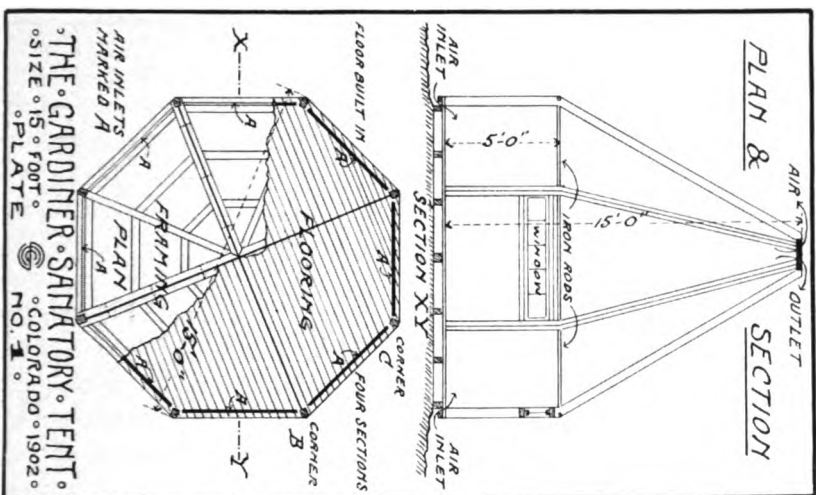
Such a method I believe I have devised by the use of my Sanatory Tent, which has been the result of considerable study and much practical experience during the last ten years. It seems to be a strong impression in most lay, and some professional, minds that if the windows of an ordinary room are opened wide, all the conditions of ventilation are then sufficient for the pulmonary invalid. This is a great mistake, and has undoubtedly caused many unnecessary deaths. Window ventilation of a room does not cause as rapid an air interchange as it should, for several reasons. If such a window faces the wind the air is forced into the room, but vitiated air is not forced out; while, if the wind is from the other direction or blowing away from the open window, the air interchange depends entirely upon suction. If two windows are used opposite each other a strong draught is created, while still the corners of the room collect stagnant air.

On a piazza, conditions are more favorable; but even here much remains to be desired. If the piazza has a roof and side curtains, these curtains have to be lowered in stormy or windy weather; there is no outlet in the roof for foul air to escape, and

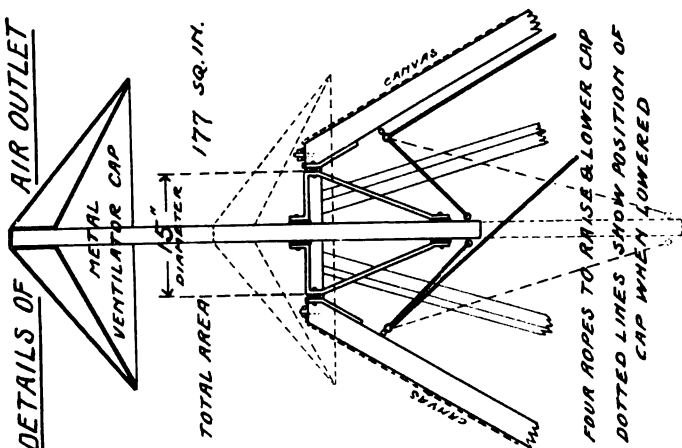
we are practically imitating the bad features of a room under the impression that we are living out-doors. This does not apply to every porch or piazza, but is true of many. On open porches, such as invalids sit upon during the daytime, the difficulty is that they are too light, windy, or cold, and if a rest is needed, or if on any pretext the house is entered, the temptation to be warm and comfortable is so hard to resist that much valuable time is lost.

These defects of room and piazza ventilation, of course, are obviated to a large extent in properly equipped sanatoria, and the results of these institutions in curing tuberculosis leave nothing to be desired. But a very small percentage of tubercular cases are ever treated in sanatoria; and I am speaking of that vast number of invalids who are trying to get well under the direction of a physician in their own homes or in boarding-houses or hotels at some health resort. Such cases we all know are very hard to control. In a sort of perfunctory way we tell them to live out-doors and be in the open air as much as possible, and the actual result is, that out of twenty-four hours about four to six are spent on the piazza and the rest in-doors. We ought to make a strong effort to utilize every hour of the twenty-four. Sleeping or awake, such patients should constantly inhale fresh out-door air. The only way to achieve this is to arrange so that they will take it unconsciously and not as a routine task to be learned like a difficult lesson. By far the best way in climates at all favorable, in my opinion, is to have them live in a sanatory tent, where they are surrounded by their comforts, where they can go to bed or sit up, where they do not have to depend on opening any windows for good fresh air, and where, if they are cold, they do not have to be loaded down by furs, with a hot bottle between their feet, but can live in a temperature of 60° or 70° F., with the perfect assurance that the air interchange is as rapid as if they were actually out-doors. This, to me, is a very essential point, and I have found that in Colorado it is practicable, and cures cases of tuberculosis that were doing badly when living in rooms and sitting on porches daily.

The invention of the sanatory tent was a slow process of evolution from a primitive and simple form which I copied from the Ute Indians. I noticed that in their "tepees" of skin the ventilation was nearly ideal. The reason for this was, they used a conical tent with a hole in the point of the top for the escape of

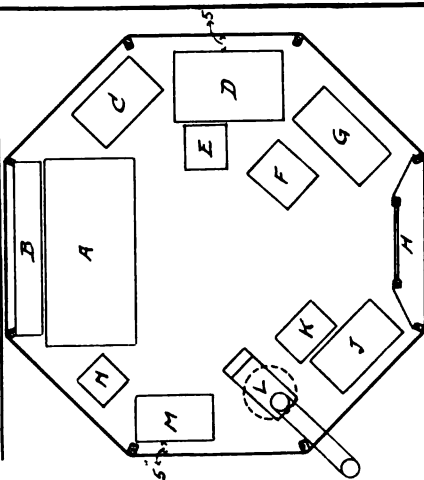


DETAILS OF AIR OUTLET



THE GARDINER SANATORY TENT.
 SIZE 15' 0" FOOT. COLORADO 1902.
 PLATE NO. 3.

FLOOR PLAN WITH FURNITURE



A - BED - 3'0" x 6'3"
 B - SHELF - 10'8" WIDE - WITH CURTAINS & HOOKS.
 C - TABLE - 2'8" x 3'6"
 D - TABLE - 1'6" x 1'6"
 E - ARM-CHAIR - 1'6" x 2'0"
 F - BUREAU - 1'7" x 3'2" - STANDING 8" FROM WALL ON ACCOUNT OF HEIGHT
 G - TRUNK - 1'7" x 2'10"
 H - WOOD-BOX - 1'3" x 1'9"
 I - WOOD-STOVE - 1'0" x 2'6" - DOTTED LINE SHOWS CYLINDER COAL-STOVE
 J - WASH-STAND - 1'6" x 2'6"
 K - CAMP-STOOL - 1'3" x 1'3"

THE GARDINER SANATORY TENT.
 SIZE 15' 0" FOOT. COLORADO 1902.
 PLATE NO. 4.

smoke and plenty of space around the lower edge, where the tent rests upon uneven ground, for air to enter it. In this way the "tepee" acts like an inhabitable chimney or fireplace, and, although in such an Indian home there are, of course, atrocious odors of all kinds, I have noticed that the actual ventilation is much better than in ordinary tents generally used by invalids.

Taking this hint from the Indians, I constructed a tent of canvas built like an Indian "tepee," which answered the purpose fairly well. I next used a circular floor with an open space all about the edge for the air to enter, still retaining the opening at the top of the tent for the heated and bad air to pass out. My tent, as now made, is of dark khaki twelve-ounce duck, stretched over a six-sided framework of wood, without any centre pole and without pegs and guy-ropes, so that it stands firm, like a house. The floor is raised eight inches from the ground, and is in six sections, so that it can be easily moved. The lower edge of the wall is fastened several inches below the floor, and one inch out from it all around; this is to insure at all times an inflow of air that is gradual and without draughts, since this inch space in a circular tent represents an area of five hundred and twenty square inches, and the hole in the top for overflowing air has an area of some one hundred and seventy-seven square inches. In this way the tent cannot be closed, and is ventilated automatically and constantly. In other words, this is a circular tent with the bottom of the canvas forming a circle around the wooden floor, and also extending a little below. The open space between the floors and the walls of the tent allows air to flow in at all times, while the hole in the apex, or top, allows air to flow out all the time. In this way the tent always ventilates itself day or night, whether the door is shut or not, whether the interior is heated or not, or in any weather. As the air has to turn a corner to enter the tent, it cannot come as a draught, and, as it passes in through all the inch space encircling the floor, it enters slowly and without force, being evenly distributed, but coming through, collectively, a large area. There are also small shutters, so constructed that they can partially close the opening from within the tent in case of very high winds. The opening at the top of the tent is covered by a zinc cone, which can be controlled by pulleys and rope within the tent, in stormy weather being drawn to within an inch of the tent roof.

My tents are heated by central draught circular stoves, which burn either wood or coal, and can be so regulated as to keep a good fire without care for ten hours. Even in zero weather the tent can be kept perfectly comfortable to dress or undress in, or to sit still and read. The stove is of such size as thoroughly to warm the tent under any conditions, and at the same time it is impossible to overheat the air or interfere with ventilation. The more heat used, the greater the displacement of heated air upward, and a more rapid interchange of air at once occurs. As the heated air can escape at the top, the fresh air can always enter at the bottom of the tent. This is automatic, and is not under the control of the invalid. These facts I wish to emphasize, as they are of very great importance. The average invalid has an aversion to ventilation, and will shut windows and in other ways embarrass the inflow of fresh air. In the sanatory tent it is impossible to do this, as it is ventilated automatically. The stove-pipe passes out through the wall of the tent and by an elbow-joint is carried upward to the height of the tent. It should be made of galvanized iron, and can be guyed in place. In order to insure good draught, and, in burning wood, to lessen danger from sparks, it must not be below the top of the tent.

A small window which does not open is used in these tents. It is placed horizontally and is one foot by six feet.

It is most essential to have this tent furnished as completely and as comfortably as a bedroom. A proper bedstead (preferably of iron) with a thick mattress and plenty of covers; a shelf with hanging curtains on both sides and ends, for hanging clothes; a washstand and bureau; if possible, a commode; a box for coal and wood; a shelf for bottles and one for books; one or two good rugs on the floor; a steamer-chair; an arm-chair, and a table for writing. These need not be expensive, but simple and strong. A scrap-basket, a broom, a dust-pan, a hammer and some nails, and a small saw frequently come in useful. In some cases I add a little zinc-lined box having two compartments, one of which is filled with ice and the other used to keep milk, eggs, etc., cool. The lighting of the tent, if possible, should be by an electric wire taken from a near-by house; there might also be an electric bell to ring in the house.

In addition to the furniture belonging to the tent, I generally have made, to use outside, an awning, twelve feet by ten feet,

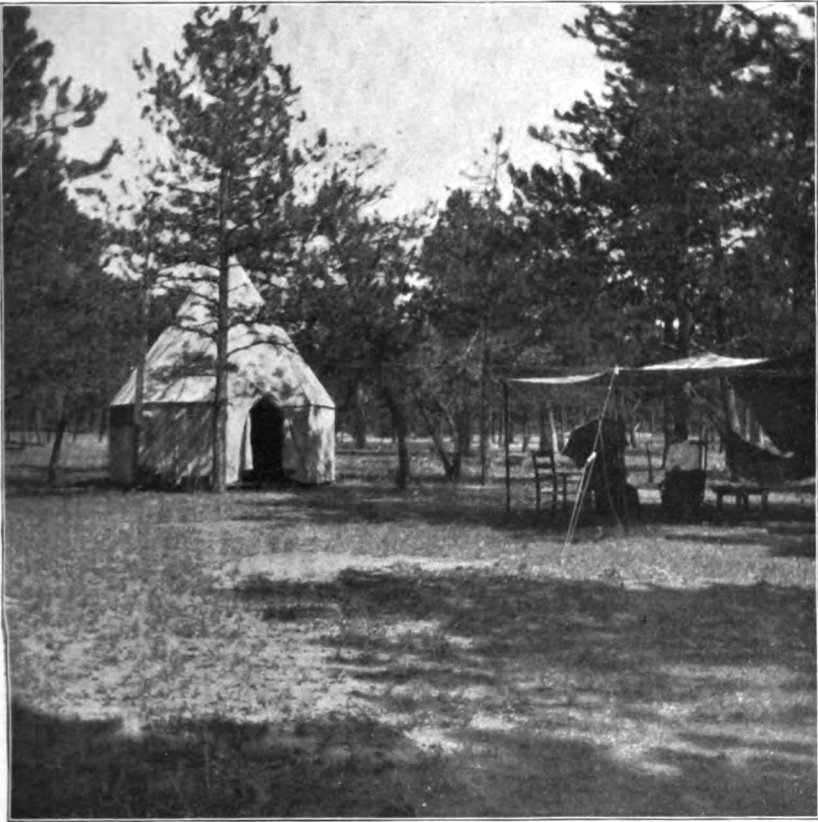


FIG. 5.—Exterior of a Dr. Gardiner's sanatory tent situated in the pines, with out-door awning and hammock.

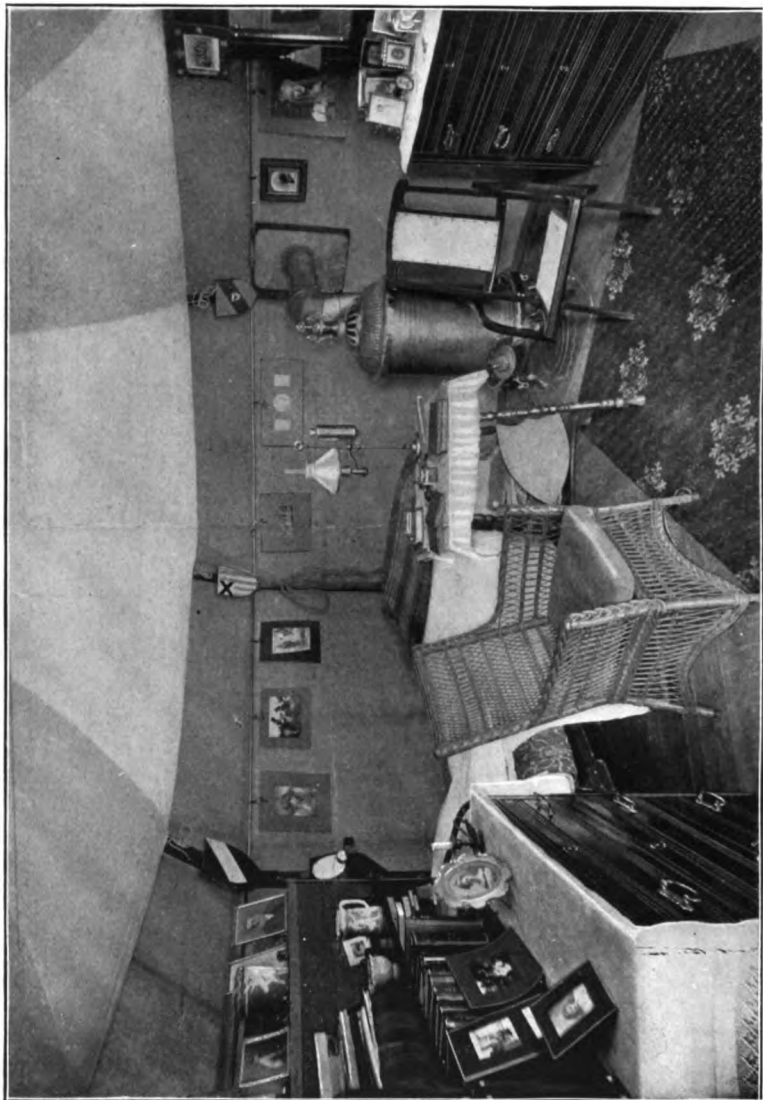


FIG. 6.—Interior of tent furnished as an ordinary room.

stretched upon four uprights, under which a hammock, chairs, and a table can be placed.

The tent is made of dark-brown duck, which does away with the white glare of light in the morning so disagreeable in most tents. The floor of the tent is about eight inches from the ground, so there is very little fear of dampness. It is, of course, more comfortable and practical for an invalid to live in this tent during the winter in a climatic dry belt such as Colorado, Mexico, Arizona, some parts of California, etc., but they have been used with success in Massachusetts, Oregon, New York, and probably in many other places.

The inherent idea common to many minds, that during the cold months living in a tent must necessarily be a hardship, is difficult to overcome. A tent generally brings up the picture in one's mind of cold, dust, wet, flies, glaring light, and, in general, the usual experiences one has on a camping trip. But in a sanatory tent, especially if any degree of common sense and care is used to have the tent in the shade during the summer, these discomforts are not felt, and invalids assure me that they are quite as comfortable as in a house. I know many invalids living in the average boarding-house room at health resorts who are certainly not as comfortable as they would be in the sanatory tent, either day or night. It is furthermore not a theory, but a proved fact, this sanatory tent having been in actual use for two or three years and under different conditions of climate. Not only have incipient cases done well, but those far advanced with cavities have had fever diminished and general improvement occur. In several instances these patients had been living in ordinary tents from a year to two years without benefit, and after living in a sanatory tent have, within a short time, improved in weight and strength.

The ventilating system of the sanatory tent construction can also be utilized in a building made entirely of wood. A small six-sided or circular house could be made of timbers and shingles, with a sharply pitched roof and an opening in the top to allow of out-flowing air, while the floor of such a building could be so constructed as to leave an open space of one inch all around inside the walls for fresh air to flow in constantly. Such a building could also be heated by an open fireplace or stove, or steam or hot water could be conveyed to it. Such a house could be comfortably furnished and have doors and windows, and, by utilizing all the sana-

tory features of the tent, would, in addition, be much more durable, and possibly be available for many invalids in damp climates during the winter months who could not be induced to live in a tent of any kind. Also, these sanatory tents, or, if preferred, sanatory tent houses, can be used as part of a general sanatorium; a main building being constructed as a heating, dining, and administration building, which could be surrounded by those tents. In this way they would be as practical as the present cottage system at far less expense, and patients living in them could be assured of a supply of out-door air, which in purity and quantity would far excel that obtained in the ordinary sanatorium cottages.¹

¹ The actual construction of these tents can be much better understood from the four drawings and the two figures which are here shown than by any description. The view of the tent interior represents a dome of canvas which divides the tent into two parts, but allows of wide space at the edge of the dome for air to pass upward by the wall of the tent. This economizes heat and is used in very cold weather. I wish to acknowledge my indebtedness to Mr. George Southard for kindly making the sectional drawings for the construction of the tent and for taking the photographs.

THE TREATMENT OF CHRONIC GASTRIC CATARRH.

CLINICAL LECTURE DELIVERED AT THE NEW YORK SCHOOL OF CLINICAL MEDICINE.

BY HEINRICH STERN, PH.D., M.D.,

Professor of Internal Medicine at the New York School of Clinical Medicine;
Visiting Physician to the Red Cross, St. Elizabeth's, and Metropolitan
Hospitals of New York City.

GENTLEMEN,—I do not consider the term gastric catarrh a good designation for the slight hyperæmia of the gastric mucosa unaccompanied by hypersecretion of mucus, which is so often spoken of as catarrh of the stomach. The process is often characterized by progressive atrophic changes, but without mucous exudate, and is, in most instances, not a catarrhal inflammation at all. Personally, I consider that some such designation as chronic gastric blennosis, denoting any chronic affection of the gastric mucous membrane, without regard to the amount of mucous exudate that may be present, is better suited to serve as a generic appellation for the various involvements of the gastric mucosa, which, with more or less propriety, are now classed together under the name chronic gastric catarrh.

There are three types of disease which may very easily be separated from the general group, each one of them having definitely recognizable characteristics and requiring special treatment for the relief of symptoms. These three forms I would call simple hyperæmic blennosis, exudative blennosis, and atrophic blennosis.

The three cases which I present to you to-day are representatives of these three typical forms of chronic gastric blennosis, or gastric catarrh.

The first case is a young man, nineteen years old. He is a native of Russia, and arrived in this country but recently. He found employment in a delicatessen store, where he had easy access to all kinds of pickled vegetables, meats, fish, etc. These highly-seasoned foods, which are mostly eaten without further preparation, were indulged in by this young man, and they have undoubtedly given origin to his present affection. He complains of loss of appe-

tite and of epigastric uneasiness and fulness, especially about two or three hours after the ingestion of food. He is constantly troubled with eructations of gas,—his stomach, indeed, is occasionally bloated to such an extent that, by its pressure upon the heart, pulsation becomes irregular and increased in frequency, and he suffers from shortness of breath. It was this set of symptoms and the fear of heart-trouble which first induced him to seek medical aid. He also suffers from constipation, and has not had a natural evacuation of the bowels for some months.

The physical examination reveals nothing except gaseous distention of the stomach and tenderness upon pressure in the pyloric region.

Clinically, this type of chronic gastric catarrh differs from other forms of indigestion by its chronicity only.

The second case is a woman, forty-six years old. Her affection belongs to the exudative variety,—that is, she is suffering from real gastric catarrh. The condition is a rather far advanced one; the patient, as you see, is very much emaciated and anæmic. She is easily excited, and complains of severe pains in the head, between the shoulder-blades, over the epigastrium, and in the lower extremities. She also exhibits all the symptoms which were found in the first case presented to-day; but, as a matter of course, they occur in a far more aggravated form. She vomits every day, before as well as after meals. The ejected material often contains bile. Mucus is always present in large quantities.

You will notice that the tongue is covered with a dense dirty-white coating. Putting the patient on the examination-table and removing the clothes from chest and abdomen, you will find, by looking down from the head of the patient, keeping your eyes on a level with the clavicles, that the region of the stomach is very prominent. Indeed, if you look very closely, you can make out almost the whole contour of the organ. The greater curvature is decidedly inflated. The pyloric end is also readily distinguishable. Percussing the stomach, and marking the area of tympany thus found with the blue pencil, you see how closely these marks tally with the outlines observed by inspection.

Pressure upon the pylorus seems to be quite painful. Pressing upon the whole organ brings on anxiety and shortness of breath.

The third case is a man, forty-two years old. He has had

gastric catarrh—that is, exudative catarrh—for more than twenty years. He himself ascribes the chronicity of his pathological state to the large amounts of tea consumed by him. Tea was at times his only nourishment. You see that the patient has greatly wasted away. There is a sickening odor emanating from him. His appearance is cachectic, and if the history of his case did not so clearly point to atrophic gastric catarrh, we would be sure to surmise the presence of a cancerous condition.

The physical examination shows the stomach decidedly diminished in size. Almost the whole organ can be felt through the integument and muscles. It appears very hard and the walls seem enormously thickened. This is especially noticeable towards the right side, in the region of the pylorus.

Proceeding now to analyze the contents of the stomach, we first perceive their peculiar brownish coloration. This is very likely caused by food which has remained in the stomach for a long period. It is not due to blood or to any of its decomposition products. You also see that the amount of juice which we have withdrawn is very small, not amounting to more than twenty cubic centimetres. The total percentage of acidity is fifteen. Hydrochloric acid is found only in traces. The greater part of the acidity, you perceive, is caused by the presence of lactic acid. Here we evidently have to deal with the so-called atrophic gastric catarrh, or anadenia of the stomach. The consideration of these three cases will carry us over one of the most interesting and important chapters in clinical medicine.

In simple hyperæmic blennosis the anatomical and physiological changes are usually slight. The surface membrane seems somewhat thicker and less smooth than in the normal state. The motor power of the stomach is also diminished to some degree. Hence, retention of food beyond the usual periods of gastric digestion, and, in almost every instance, hyperfermentation, with production of lactic, acetic, and butyric acids and excessive formation of gases. The amount of mucus in the empty stomach affected with hyperæmic blennosis is normal, or even subnormal.

Clinically, simple, chronic hyperæmic blennosis exhibits a picture not differing essentially from that of other types of indigestion. The appetite is diminished in many cases, but not in all; there is epigastric uneasiness and fulness, particularly some time

after ingestion of food, and eructations of gas are the rule; the burning sensation commonly called heart-burn, arising from gastric hyperfermentation and putrefaction, is frequently met with; there is apt to be nausea and the eructation of bitter, disagreeably tasting liquid, which causes pharyngeal, buccal, and oral irritation. This is not uncommon, especially when lying down. Dulness, cephalalgia, indisposition to mental work, and somnolence are almost always concomitant with gastric disorder. The tongue in this type of blennosis does not, as a rule, exhibit any special characteristics.

The anatomical and physiological changes in chronic exudative blennosis are more pronounced. The mucosa is decidedly thickened and toughened, especially in the pyloric region. It is vascular, and its normal pinkish or pale yellow color is changed to hyperæmic red or brown, or, in most instances, to gray. Not infrequently the pyloric portion is found slate-colored, and once in a while it appears very dark. These alterations are due to capillary destruction and subsequent pigmentation. The surface, invariably covered with grayish-white or colorless tough and ductile mucus, appears uneven, as a general rule, and is often spoken of as warty. This characteristic papillary appearance of the inflamed gastric mucosa, the "surface mamelonnée," or benippled surface, is most pronounced near the pylorus.

Involvement of the pyloric submucous and muscular coats in the hypertrophic process may occasion narrowing of the orifice, which in turn may result in gastrectasis. The main characteristic of exudative blennosis is the excessive secretion of mucus. This catarrhal phenomenon, of constant occurrence in the empty as well as in the full stomach, stamps this type of blennosis as the true chronic gastric catarrh. The term, if used at all, should be employed to designate only this particular condition. The total acidity of the gastric contents, in the majority of instances, does not seem so small as is commonly supposed. The acidity is due, in some measure, to the formation of lactic, acetic, and butyric acids, and also to the presence, although usually in diminished quantity, of free hydrochloric acid. In certain cases the amount of free hydrochloric acid is undoubtedly greatly reduced, but I have not found it entirely absent in a single instance of chronic exudative blennosis. On the other hand, excess of hydrochloric acid is not uncommon. This, as might be expected, interferes with the proper digestion of carbohydrate

substances. However, in such cases the digestive power of the stomach is less impaired than is its motor mechanism. The digestive ferments are generally, though not invariably, found in diminished quantity. In some instances it is extremely difficult to recognize their presence at all. Motor insufficiency of the organ is much more in evidence than in simple hyperæmic blennosis. The syndrome of this type of the affection is also present, but in a considerably aggravated form. Nausea is of common occurrence; anorexia is more marked than in hyperæmic blennosis. Desire and distaste for certain articles of food are often dependent entirely on idiosyncrasy. Vomiting occurs but rarely in some cases; in others it is almost a permanent factor. Ordinarily the vomit consists of the remains of food and of mucus with which biliary fluid is occasionally mixed. However, vomiting is not always dependent upon the presence of food in the stomach. In the chronic exudative blennosis of alcoholics, for instance, there exists often a tendency to vomit in the morning before breakfast, in which case the egested material consists of nothing but large masses of viscid mucus,—the so-called “water-brash.” Gastrorrhœa—the ejection from the stomach of quite large quantities of watery liquid—is often a symptom of a deranged state of nervous mechanism, and occurs mostly in chronic blennosis of the neurasthenic type. The appearance of the tongue in chronic exudative blennosis is often characteristic of the gastric condition. In advanced cases it is generally large, broad, flabby, and furred mostly with a thick dirty-white or coffee-brown coating. This is particularly the case when the catarrhal condition has involved the oral mucosa. On scraping off the coating, the papillæ are seen to be unusually prominent. In certain cases the tongue appears small, red, and uncoated, and with enlarged papillæ on the tips and edges.

In atrophic blennosis, the third form of chronic gastric inflammation, very decisive anatomical and physiological changes occur. The gastric glands and tubules undergo sclerotic degeneration,—their caliber diminishing in the ratio in which the epithelium vanishes. Ultimately the surface of the mucosa may become almost smooth. Interglandular tissues and submucous and muscular coats gradually become involved in the atrophic process. The entire gastric wall may attain enormous thickness, and the capacity of the stomach in the large majority of cases becomes greatly reduced.

In atrophic blennosis there is almost always more or less achylia. The fasting stomach contains, if anything at all, but mere traces of juice. Directly after the ingestion of food no acid whatsoever can be detected. A little later, acidity, due to lactic, acetic, and butyric fermentation, may ensue. Concurrently with the absence of hydrochloric acid there is also a lack of digestive ferments.

The degree of motor insufficiency is very striking, and solid food is retained in the stomach for hours. If it is not vomited, it slowly passes through the pylorus, after which it will undergo intestinal digestion. This may suffice to keep up nutrition for a certain period, but progressive weakness and emaciation are inevitable. Complete anorexia, offensive breath, severe gastric pain, and anæmia resembling the pernicious variety are of very frequent occurrence.

The chronic gastric blennoses are hardly ever independent affections. They are nearly always associated with other disorders, or are but the consequence of other pathological conditions. Any of the lighter forms of gastric blennosis may depend etiologically upon a previous cardiac affection; chronic gastric catarrh may arise from circulatory disturbances, more especially in the stomach itself, or from obstructions in the portal circulation. It is often found together with chronic affections of the bronchi and lungs. Almost every case of phthisis pulmonum which I have observed in dispensary practice was accompanied by some form of chronic gastric blennosis.

There is hardly any wasting disease, any cachectic condition, which sooner or later is not followed by gastric blennosis. Thus it is seen that chronic gastric blennosis is very often but a symptom, a mere consequence of an underlying disorder. The successful treatment, or the removal of the causative factor of the gastric condition, often results in its complete amelioration. To treat or to remove the cause, whenever practicable, should therefore always remain our foremost object.

Diet.—Since errors in diet, and particularly over-indulgence in stimulants, alcoholic as well as alkaloidal, alone or together with other causes, stand at the foundation of many cases of congestive or catarrhal blennosis, their dietary treatment is of special importance. There are, however, no gastric diseases for which less definite dietary rules may be laid down than for these affections,

that is, there are no gastric affections in the dietary treatment of which we have to individualize to such a degree as in the two lighter forms of mucous inflammation.

In atrophic blennosis, on the other hand, there is not much gastric individualization left, and so we can have recourse to an absolute regimen.

The general dietary treatment of hyperæmic and exudative blennosis consists mainly in the complete or partial exclusion of such ingesta, solid as well as liquid, as are known to have given rise to gastric symptoms. The mere withdrawal of liquors, or of tea or coffee, in most instances, is speedily followed by subsidence, or, as in some cases, by even complete disappearance of the gastric difficulty. Similar results may be obtained if the patient abstains from certain dishes, or from late suppers, if these have proved to be the main cause of the chronic inflammatory processes in the stomach.

In my experience chemical examination of the gastric juice or contents of the stomach in chronic gastritis does not furnish a trustworthy clue as to the real dietary needs of the patient.

If a regimen is to be prescribed for chronic gastric catarrh, the chemistry of the gastric contents, provided there is not the total absence of the gastric juice which occurs in atrophy of the organ, should not play the important rôle which some clinicians have assigned to it. There are other factors at least as potent to be taken into consideration when a diet is to be agreed upon, principal among which is motor insufficiency. We may diagnose hypochlorhydria, a very frequent occurrence in chronic gastric catarrh, and still the patient may have less difficulty in digesting a meal rich in protein matter than one in which farinaceous substances predominate. Clinical experience alone, and not theory, should guide us when we are about to devise a dietary for those afflicted with chronic gastritis.

As stated before, no definite rules of diet for all cases of a certain type can be given. Loss of appetite, a common occurrence in gastric blennoses, encourages the taking of but small quantities of nutriment, and is a useful ally in the dietary treatment. Besides this, I have found it best to begin with a single type of food, and to administer it irrespective of the usual meal-hours, as infrequently or as often as it seemed necessary. If agreeable to the patient, he

may take it in gradually increasing amounts; if not, it should be withdrawn and another article of food substituted. A greater variety of ingesta may be permitted in the ratio in which the condition improves, but more than one kind of food, and this in small amounts, should never be added at a time.

I have found the following foodstuffs, enumerated in accordance with their general usefulness, well adapted for the majority of cases of hyperæmic and exudative blennosis.

Milk, the least irritating of all nutriment, is best suited at the outset of the dietary treatment. A pure high-grade quality should be insisted upon. Used unskimmed and raw, it is followed by excellent results in most instances. In winter it is advisable to take the chill from it by placing the bottle containing it in a little hot water. In advanced cases it should be sipped with a spoon, and the quantity for a person weighing sixty kilogrammes should be restricted to two or three litres during the twenty-four hours.

In instances complicated by gastric atony and dilatation a similar course should be pursued. A milk-regimen is not always well borne during the first few days. The addition of sodium bicarbonate, or lime-water, or mineral water, diminishes its tendency to acid fermentation. It should never be employed alone in the lighter form of the disease for protracted periods; in the atrophic variety it may serve as a permanent nutriment, especially if predigested. Occasionally a milk-regimen may bring on persistent indigestion and vomiting. In such cases the milk should be withdrawn, to be replaced by koumiss or matzoon.

In the pre-atrophic stages of gastric catarrh meat of proper quality, suitably prepared, is the foodstuff mostly to be depended upon in the long run. Beef, veal, lamb-chops, and chicken may be partaken of. The meat should be tender and lean. Scraped or chopped beef, to which some salt and pepper may be added, eaten in the raw condition, is probably easier of digestion than any other nutriment. Even in cases where hypochlorhydria is an established fact, raw beef hardly ever causes gastric difficulty. Among the solid foods, it seems the one best adapted when pronounced motor insufficiency concurs with chronic catarrh of the stomach. All other kinds of meat should be roasted or broiled. Gravies, if at all permitted, should not contain fatty material, nor should they be highly seasoned. In beginning, the regimen and daily allow-

ance of meat for the individual weighing sixty kilogrammes should not exceed fifty grammes. From twenty to fifty grammes may be added every day until the total diurnal ration amounts to five hundred grammes. If meat preponderates in the diet for a protracted period, steps must be taken to avert untoward results. This is best accomplished by the ingestion of alkaline waters, not carbonated artificially.

Solutions of pure gelatin seasoned with salt and pepper are not only far superior in nutritive quality to bouillon and clear soups, but pass the stomach rapidly without causing untoward symptoms. Jellies, etc., of the same substance, flavored with some lemon- or grape-juice, possess great nutritive and body-albumin-saving properties. The gelatinous preparations may be used to advantage even in the atrophic form of the affection.

In cases of hyperæmic and exudative blennosis, where the secretion of hydrochloric acid does not deviate from the normal, eggs serve as an ideal food. They should be eaten either raw or soft boiled. In the latter case they must not be kept longer than from forty-five to sixty seconds in the boiling water. If raw or soft-boiled eggs are repulsive to the patient, and if, for the time being, no food seems to agree with him, hard-boiled eggs, finely divided, may serve a useful purpose. If the catarrhal condition is accompanied by pronounced achylia gastrica, eggs, probably more on account of their fatty than their albuminous constituents, frequently cause symptoms of indigestion.

When they contain but small amounts of fat, fish and sea-foods are generally well borne by the catarrhal stomach. Best suited for this condition are: pike, trout, perch, sole, haddock, and oysters. The latter should be partaken of on the half-shell only; as condiments horseradish or lemon may be used. Fish should be broiled or boiled and served without dressing. A slice of lemon or some mustard may be substituted for the latter. The gelatinous substances contained in fish render it a very valuable nutriment.

In many cases of non-atrophic gastritis, fat is permissible in the form of eggs. Cream in its usual percentage in milk hardly ever causes gastric discomfort. Some individuals with chronic catarrhal blennosis exhibit a moderate tolerance of fat. Egg and milk fat should always be given the preference. In pyloric obstruction, and when pronounced motor disturbance is present, the

formation of butyric acid should be prevented by the complete exclusion of all fatty matters from the nutriment. Whenever fat can be employed, it serves as a most useful addition to the diet on account of its high caloric value.

Farinaceous material lodging in the stomach for any length of time is apt to undergo lactic and acetic acid fermentation. As the motility of the organ is always more or less interfered with when a catarrhal condition supervenes, starchy substances, *a priori*, should be eliminated from the dietary unless there is sufficient proof that they may be tolerated. Vegetables and other more bulky material must be excluded. This should be the case not only when gastrectasis is present, but in every single instance of chronic gastric blennosis. The only carbohydrates which I permit are farina, rice, toasted wheaten bread, and small amounts of sugar. Sometimes they can be added to the foods after two weeks of a strict milk-regimen, at other times months of dieting are necessary before they may be included among the permissible nutriments.

Farina and rice should be well boiled in water. They should be "boiled in" until indeed the lowest stratum is somewhat burned and sticks to the pot. Stale wheaten bread should be utilized in making toast. It is best to serve it well browned on both sides.

In the beginning of treatment no fruit should be allowed. Later on, especially in the absence of hyperchlorhydria, an orange at breakfast, purée of pears, or apple-sauce well strained, and baked apples may be included in the dietary.

Apart from the water contained in milk and the diverse nutrient solutions, an additional amount of pure drinking water should always be ordered in uncomplicated cases. The exact quantity has to be adjusted to the needs of the individual case, but it stands to reason that moderate amounts only should be prescribed. When gastropnoia or gastrectasis are present, the amount of all the liquids has to be reduced to the smallest possible limit. The drinking water should be absolutely pure and not cooler than 10° C. (50° F.). In place of it natural mineral waters should be used. Waters artificially charged with carbon dioxide always contain an excess of gas, and are, therefore, not suitable in chronic gastric blennosis.

Alcoholic and malted beverages, even if they do not stand at the foundation of the pathological condition, must be positively ex-

cluded from the dietary of chronic gastric catarrh. Apart from their toxic and otherwise deleterious effects upon the organism, we must recall that alcohol increases the tendency to connective-tissue formation. This at once precludes its employment in all conditions liable to be complicated by atrophic changes.

As long as other unnecessary substances of diet—namely, spices, flavors, and non-alcoholic stimulants—are not inducers of chronic gastric blennosis, they may be added to the dietary after some time, in moderation, however, only. In some instances I find it necessary to forbid tea or coffee entirely. While condiments and flavors may be omitted by the patient for a certain time, their prohibition for protracted periods is neither practical nor heeded, as a general rule.

The diet in atrophic gastric blennosis is very important. Milk and milk-preparations often pass the atrophic stomach without molesting it in any degree. In case the milk does not agree, proper medication should be employed to make it acceptable to the gastric organ. However, if these means fail, the milk should be predigested before it is partaken of. Artificially digested in the usual way, it is often borne well for months and even years. Next to milk in general value I have found gelatin in aqueous solution. This seems to pass through the pylorus in a very short time, and its ingestion, I think, is followed by at least as satisfactory results as the use of many of the soluble liquid semidigested foods found on the market, though many of these preparations, however, serve a good purpose. When there is absolutely achylia gastrica, it may be necessary to admit sugars and starches into the dietary. In such instances care should be taken to prevent hyperfermentation in the stomach. This is best accomplished by the administration of a gastric antifermentative.

In far advanced cases rectal and subcutaneous alimentation have often to be resorted to.

The treatment of these three conditions consists in the regulation of the diet, of lavage and irrigation, the use of drugs, and physical therapeutic measures.

Lavage of the stomach in various modifications is a most efficient treatment in all forms of chronic blennosis, but it is the means *par excellence* of alleviating or curing exudative blennosis, the true catarrhal form of chronic gastritis.

The stomach-tube is nowadays known to every practitioner of
Vol. IV. Ser. 12—2

medicine. It should be of soft rubber, its length amounting to about one hundred and thirty-five centimetres, and its lumen from No. 26 to 32, of the French scale. The extreme end of the tube which is to be introduced into the stomach may be open, and in addition there may be one or more perforations in its sides. Some tubes are closed at the terminal extremity and fenestrated in the sides only. For the withdrawal of the contents of the stomach, after a test-meal, for instance, I prefer the tube open at its extreme end. For washing out the stomach, the one with lateral fenestræ only possesses certain advantages of which I shall speak presently. When the tube is introduced so that a mark on it (generally placed at about sixty centimetres from its perforated end) is in proximity to the lips, then the lower end has well entered the cavity of the stomach.

The technic of lavage is so well understood that there is no need to dwell on that subject. To one point, however, I want to draw attention. If the tube is admitted beyond the cardiac orifice, so that its perforated part is found within the gastric cavity, the cleansing fluid when passing down will come but very little into contact with the gastric walls. This is especially the case when the extreme end of the tube is open. The solution thus introduced will loosen some of the exudate, it is true, but this is accomplished by its solvent properties merely when accumulating in the cavity. Hence, by pursuing this method, but a small portion of the stomach-wall receives direct irrigation, and, as natural, an amount of mucus will remain behind. If, however, a tube closed at the extreme end with openings in the sides is introduced, so that the perforated part is situated within the cardiac orifice, the irrigation of the stomach-walls can be performed in a most successful manner. The irrigating fluid entering from the œsophageal opening, while drizzling down the walls of the empty stomach, loosens and removes the viscid mucus and stimulates the surface. As soon as the stomach is filled to a certain extent, the tube is pushed farther down to withdraw the irrigating fluid in which the mucus is kept suspended.

This method, which was first introduced by Richter, of Munster, permits direct and complete irrigation of the stomach-walls. With some practice the attendant will soon learn just how far to admit the tube. Besides, if the water enters at the cardiac orifice, the patient on being questioned will state that it drizzles down the stomach-walls, a sensation not experienced by him if lavage is per-



FIG. 1.—Dr. Stern's apparatus for irrigation of the stomach.



FIG. 2.—Method of using Dr. Stern's apparatus in irrigation of the stomach.

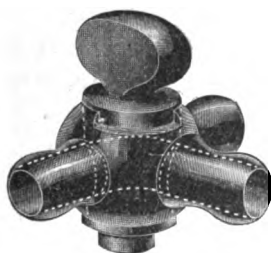
formed in the usual manner. "Wall irrigation," a designation appropriate for this method, is also well adapted when gastric dilatation and atony complicate catarrhal blennosis.

Continued irrigation not only cleanses the stomach more thoroughly than does the simple washing with one or two litres of water, but it also tends to impart tonicity to the mucosa and the deeper lying tissues. It acts, so to speak, as a species of internal massage, equalizing the circulation and stimulating motor power and glandular activity of the stomach.

I have devised an apparatus for convenient and rational irrigation of the stomach. It does away with the necessity for a funnel and pitcher, and obviates inexactness, spilling of fluids, and waste of strength.

The apparatus can be understood from a glance at the illustrations (Figs. 1 and 2). To a stand five feet high, which by means of a crank mechanism can be readily raised to seven feet, two or more percolators of the capacity of eight litres each are fixed in such a manner that their cleansing, removal, etc., are easy. A bowl made of enamelled iron, like the rest of the stand, is provided for the reception of the stomach-tube. A bucket graduated in litres on the inner side and designed to receive the returning irrigating fluid is also attached to the stand. In the apparatus here described a

FIG. 3.



black rubber hose of the diameter of the average stomach-tube is connected with the two percolators, each having a graduation of from five hundred to eight thousand centimetres, by means of a glass V and two short rubber tubes. Each of the latter communicates directly with one of the percolators, and is provided with a cut-off. At the other end of the hose, which is of suitable length, there is a double-flow, hard rubber faucet (Fig. 3). The caliber of

the faucet permitting entrance of the irrigation-fluid is somewhat smaller than that of the cannula for the egress of the same. Thus the cannula, which is of the same diameter as the average stomach-tube, will not be clogged by the return-flow carrying mucus and débris. A glance at the diagram of the faucet on page 19 shows that there is no angle interfering with the outflow. Attached to the faucet, to the in- and out-flow portions respectively, are the stomach-tube proper and the waste-tube, which latter leads to the graduated bucket.

Before filling the percolators with the irrigating fluid or fluids, care should be taken that cut-offs and faucet are tightly closed. The various percolators may contain the same solution; however, it may be found best to employ different irrigating media in strength as well as in character. If a large amount of but one kind of irrigation-fluid is to be employed, as, for instance, a two per cent. solution of sodium bicarbonate, the two percolators may be filled with it at once, and as soon as one is emptied, the contents of the other may be admitted into the stomach by simply loosening the cut-off.

The stomach-tube is introduced in the usual manner, but care should be taken that it does not pass beyond the œsophageal orifice. This, I think, is not only the most valuable, but also the safest procedure, as it will permit irrigation, even if ulcers and other complications are present.

The irrigating current should be regulated according to the general systemic status and the specific gastric condition. Two effective means are at our disposal to accomplish this end. First, by raising or lowering the percolators with the assistance of the crank mechanism, thereby increasing or diminishing the velocity of the current; and, secondly, by regulating the inflow with the faucet. In the atrophic variety of the congestive blennosis, when ulcers and other complications are present, the current velocity should be smaller than when an exudative blennosis is dealt with.

When the stomach is almost filled with the irrigating fluid, the stop-cock should be turned in such a way that a communication is established between the stomach- and waste-tubes. This will cause a siphoning action and emptying of the stomach, as if an ordinary stomach-tube were utilized. The amount of returned irrigation-liquid is then read off from the scale in the bucket and compared

with that used from the percolators. This will show at once whether irrigation-water is retained in the stomach, or whether there was some liquid therein when irrigation was started.

As soon as the stomach is empty, the stop-cock is turned so that it shows in the same direction as the stomach-tube. This permits the inflow of irrigating solution once more and interrupts the siphoning action.

If, instead of irrigation, lavage has been performed,—that is, if the stomach-tube was introduced beyond the cardia,—then, in order to remove a greater amount of mucus and to stimulate a larger area of mucosa, it may be found necessary to change the position of the patient from time to time during the application of lavage; movements from side to side, reclining and rocking are often resorted to under these circumstances.

The stomach spring tube, devised by Rosenheim, exhibiting numerous lateral springs of one-half millimetre diameter, through which the water enters forcefully into the stomach, is well adapted for the treatment of various forms of motor insufficiency. In the treatment of exudative gastric blennosis, however, the method here outlined possesses marked advantages.

In the therapy of chronic gastric blennosis medicines do not play an unimportant rôle. We employ medicinal agents for the following purposes: 1. For cleansing the stomach and dissolving the mucus. 2. To prevent fermentative and toxic processes in the stomach. 3. To stimulate secretory and digestive activity of the gastric organ. 4. To soothe and alleviate pain and to relieve vomiting. 5. To induce functional activity of other organs involved and to promote general nutrition.

1. As to the first indication soapsuds and saline solutions are effective in detaching mucous exudate and cleansing the stomach. Sodium chloride or sodium bicarbonate in one-half of one per cent. to five per cent. solutions are frequently utilized for that purpose. Their best mode of employment is as irrigators with the stomach-tube. As a further aid in cleansing the gastric mucosa, and as a general antiseptic, I have devised the following tablet:

R Sodii boratis, 8.0 grammes (gr. xlv);
Resorcin, 1.0 gramme (gr. xv);
Menthhol, 0.75 gramme (gr. xii);
Sodii benzoatis, 5.0 grammes (gr. lxxv).

For lavage one tablet is used in one or two tumblerfuls of tepid water. The best way to administer it, however, is by irrigation, either combined with one of the salines mentioned or alone. I generally irrigate with the tablet solution after the mucus has been partly removed by a saline cleansing fluid. My apparatus for stomach-irrigation is especially well adapted to execute this treatment. The saline cleansing liquid is prepared in one, the tablet solution in another percolator. From one to three tablets for each litre of water suffice for all ordinary purposes.

2. Among the gastro-antiseptics may be enumerated: Carbolic acid (0.015-0.1 gramme), well diluted in water, or in pill form; thymol, in the same dose as carbolic acid, in pill form, with powdered soap as the excipient; ichthyol (0.15-0.75 gramme), in capsules or in water; salicylic acid and the salicylates, particularly bismuth salicylate,—this is best given in powder form or in some mucilaginous vehicle in varying doses; boric acid (0.3 to one gramme); resorcin (0.3-0.75 gramme); creosote (0.05-0.15 gramme).

The gastro-antiseptics may be prescribed alone or together with other medicinal agents.

Creosote for gastro-antiseptics I prescribe, as a rule, in one of the following formulæ:

R Creosoti (beechwood), 0.05 to 0.15 gramme (℥ $\frac{1}{2}$ to ii);
 Pulv. carbonis ligni, 0.25 gramme (gr. iv);
 Mucilaginis tragacanthæ, q. s.
 M. ft. one pill.
 Sig.—One pill half-hour before meals.

R Creosoti (beechwood), 0.15 gramme (℥ ii);
 Magnesii salicylici, 0.15 gramme (gr. ii);
 Tr. gentianæ comp.;
 Aquæ menthæ pip., ãã 4 grammes (f3i).—M.
 Sig.—Before or after meals, according to indications.

Creosote is especially valuable in the presence of flatulency and gastric pain after eating.

3. The so-called stomachics are legion. The most innocent of the gastric stimulants, those which may be employed for protracted periods, provided they do not contain too much alcohol, are the bitter tonics. These are indicated in almost every case of hyperæmic or exudative blennosis. Their action is enhanced when the

stomach is cleared of mucus and is free from food. In most cases, if given after meals, they prevent accumulation of gases and consequent discomfort. Among the vegetable drugs stimulating secretory and digestive activity we have:

Calumba, *cinchona*, *condurango*, *frasera*, *gentiana*, *hydrastis*, *nux vomica*, *piper*, *quassia*, *xanthoxylum*, and *zingiberis*.

Drugs like *calumba*, *cinchona*, or *gentiana*, if ordered uncombined with other medicines, should not be prescribed in the form of tinctures, as by their continuous use the patient acquires the alcohol habit only too readily.

I append some prescriptions which I have found of great service in the treatment of individual cases of gastric blennosis:

As a mild digestive, I often use:

R Ext. *calumbæ*, 0.05 gramme (gr. $\frac{1}{4}$);
 Ext. *rhei*, 0.15 gramme (gr. ii);
 Pulv. *piperis nigr.*, 0.1 gramme (gr. iss).
 Misce et fiat capsula, No. 1.
 Sig.—Before meals.

For anorexia, gastric atony, and flatulence:

R Ext. *collinsoniæ fluidi*, 0.5 gramme (gr. vii);
 Ext. *cinchonæ fluidi*, 1 gramme (℥xv);
 Tinct. *capsici*, 0.1 gramme (gr. iss);
 Ext. *cardamomi compos. fluidi*, 1 gramme (℥xv);
 Aquæ, q.s. ad 8 grammes (ad ℥ii).—M.
 Sig.—Half-hour before meals.

For gastric atony in the presence of ulceration:

R Decoct. *condurango*, five per cent., 50 grammes (℥iss);
 Infus. *matricariæ*, five per cent., 100 grammes (℥iii).—M.
 Sig.—Three or four times a day, before meals, warmed to 40° C. (105° F.).

Appetizer and gastric stimulant:

R Infus. *gentinæ compos.*, 15 grammes (℥ss);
 Ext. *zingiberis fluidi*, 0.5 gramme (gr. viii);
 Aquæ *menthæ piper.*, q.s. ad 30 grammes (ad ℥i).—M.
 Sig.—Half-hour before meals.

In my experience *hydrastis* has proved not only the most valuable of gastric stimulants, but also the most effective remedy for the various pathological conditions of the stomach. It reduces atonicity, modifies abnormal secretion, stimulates the secreting

functions of the gastric glands, and conduces materially to the general tone of the system.

It may be administered alone or in combination with other medicines, and in the form of extract, fluid extract, tincture, or as hydrastine, hydrastine hydrochlorate, or hydrastinine hydrochlorate.

The following stimulating, antifermentative, neutralizing, and cathartic combinations have rendered me excellent service:

Antifermentative, antacid, gastric stimulant, and cholagogue:

R Sodii bicarbonat., 0.5 gramme (gr. viii);
 Tr. nucis vomicæ, 0.3 gramme (℥v);
 Ext. rhei fluidi, 0.25 gramme (℥iv);
 Ext. hydrastis canadensis, 0.4 gramme (gr. vi);
 Aquæ menthæ piperitæ, q.s. ad 4 grammes (ʒi).—M.

Sig.—Before or after meals, according to circumstances. Shake well.

Gastric and general stimulant:

R Ext. hydrastis canadensis fluid.;
 Ext. collinsoniæ fluidi, aa 0.35 gramme (℥vi);
 Infusi gentianæ compositi, 15 grammes (ʒss).—M.
 Sig.—Half-hour before meals.

Gastro-hepatic stimulant and cathartic:

R Hydrastine, 0.05 gramme (gr. ʒ);
 Podophyllin, 0.003 gramme (gr. ʒss);
 Oleoresinæ iridis, 0.15 gramme (gr. ii).
 M. et fiat pil. No. 1.
 Sig.—One or two pills at bedtime.

Sedative drugs are not infrequently important in the treatment of chronic gastric blennosis. Most gastric sedatives afford relief of vomiting. The insoluble bismuth salts, when brought into intimate contact with the stomach-walls, will alleviate pain, provided it is not of too fervid a nature. The only rational method in which to employ any of these compounds is first to free the mucosa from exudate by irrigation and then to administer the bismuth preparation in mucilaginous suspension. If gastrodynia is more pronounced, the addition of some diluted hydrocyanic acid will usually increase the efficiency of the bismuth salts. Hydrastis, together with bismuth salicylate, will often afford prompt relief, particularly if the pain is due to atony.

Some coal-tar derivatives, bromides, iodides, cannabis indica, carbolic acid, creosote, salicylic acid and salicylates, silver-salts, alkaloids, and a great number of other medicinal agents have been employed in the various forms of the gastric blennosis for the relief of pain as well as of vomiting.

Morphine and atropine are gastric sedatives. Their action upon the secretion of gastric juice, however, differs widely. While morphine, contrary to the opinion formerly held, increases the secretion, atropine has the tendency to arrest it.

In cases of gastralgia which are due to hyperacidity or hypersecretion of gastric juice, atropine is thus the indicated remedy. If, on the other hand, gastrodynia concurs with hypersecretion or motor insufficiency, morphine will render efficient service.

The silver-salts are best administered in pill form, half an hour, or even an hour, before meal-time. Alkalies, chlorides, bromides, iodides, etc., should not be used if silver nitrate is to be taken.

The following combinations I have found very useful in chronic gastric blennosis, accompanied by gastrodynia, etc.:

For the relief of pain, ulceration, and vomiting:

R Morphinæ hydrochlorat., 0.01 gramme (gr. $\frac{1}{10}$);
 Argenti nitrat., 0.02 gramme (gr. $\frac{1}{10}$);
 Ext. gentianæ, q.s.
 M. et. ft. pil. No. 1.
 Sig.—Every three or four hours.

For the relief of neuralgic pain in the presence of chronic gastric catarrh:

R Tr. gelsemii, 0.5 gramme (℥ vii);
 Ext. hyoscyami fluid, 0.15 gramme (℥ ii);
 Spts. chloroformi, 1.5 grammes (℥ xx).—M.
 Sig.—Three to six times a day in half a tumbler of water.

As chronic gastric blennosis is very often but the expression of an underlying, more general disorder involving, to a greater or a lesser degree, other organs, it is evident that the latter, as well as the entire organism, demand stimulating tonic treatment.

Many of the special and systemic tonics may also be employed in the presence of the various forms of gastric blennosis.

Various preparations of iron, pre-eminently the albuminized or peptonized forms; strychnine; adonidin; digitalis and its preparations; dimethyl and trimethyl xanthine; strophanthus; spar-

teine; nitroglycerin, etc., all have their special indications, and, in moderate doses, are, as a general rule, well borne by the gastric organ affected with hyperæmic and exudative blennosis. In atrophic blennosis, rectal or hypodermic medication is often imperative.

Physico-therapeutics, etc.—The physical treatment of chronic gastric blennosis may consist of: Rest; walking, climbing, etc.; calisthenics; mechano-therapeutics; massage; pneumatic gymnastics; electricity; hydrotherapy, etc.

The institution of physical treatment for chronic gastric blennosis and the selection of the various measures should depend not only upon the gastric condition itself, but also upon the general systemic status of the patient.

When beginning treatment, particularly when the patient has just been put on a strict milk-regimen, rest in bed for a few days, although not always absolutely essential, will invariably exert a wholesome influence upon the gastric and systemic conditions. Cases in which the nervous system is primarily at fault are often greatly benefited by absolute rest continued for protracted periods. Patients with advanced exudative blennosis and those afflicted with gastric atrophy need much rest in bed, and this mode of treatment should always be given preference.

A certain amount of exercise is essential in the treatment of the congestive form of catarrhal blennosis. However, the kind and duration of the various exercises have to be strictly adapted to the needs of the individual case. In some instances gymnastics of the lower extremities, in others exercise of the upper extremities and the thorax are indicated. Calisthenics serve a useful purpose in many cases, particularly if executed under the direction of an experienced instructor. Mechano-therapeutics, resistance movements, etc., have their special value if cardiac disease, obesity, arthritis urica, etc., are associated with the gastric affection.

Massage of the epigastrium, in my experience, has not proved of any material value in ameliorating chronic gastric blennosis; on the contrary, in many instances, as in the presence of acute inflammatory processes, ulcerations, carcinoma, etc., epigastric massage and kindred manipulations are absolutely contraindicated. On the other hand, massage of the lower abdomen, to relieve obstipation to the cardiac region and to the limbs, will frequently prove of decided advantage.

Massage of the stomach and colon, after the introduction of certain medicated liquids, has been recommended of late. I have found, however, that with direct massage to the gastric mucosa by means of the irrigating current, as I employ it, much more can be accomplished in the way of stimulation than by any other method of massage.

Pneumatic gymnastics, by the employment of Turck's device, probably contribute to the amelioration of gastric atony accompanying chronic gastric blennosis. I have as yet very little experience with the method, but do not hesitate to recommend it on theoretical grounds.

Electricity, for general tonic treatment, often proves a potent therapeutic agent. Employed intragastrically, to promote secretion and to ameliorate myasthenia, I find it to be of indifferent value.

Hydrotherapy, in its various modifications, is of great service in many instances of chronic gastric blennosis. Application of hot packs and steam and Turkish baths undoubtedly tend to overcome achylia gastrica. Cold baths, on the other hand, while possibly invigorating the organism, apparently do not promote the secretion of gastric juice. Sitz- and foot-baths of 45° C. (112° F.) and over, by regulating the general circulation, may exhibit a curative influence upon the chronic gastric blennosis.

SOME PRACTICAL POINTS ON THE EARLY DIAGNOSIS AND TREATMENT OF MALIGNANT DISEASE OF THE LARYNX.

BY PHILIP R. W. DE SANTI, F.R.C.S.,

Surgeon. Laryngologist and Aural Surgeon to Westminster Hospital; Late Senior Assistant Surgeon to Westminster Hospital, London.

DURING the last fifteen years or more a very large amount of good work has been done in England and abroad on the subject of the early diagnosis and operative treatment of malignant disease of the larynx, and it is with these two aspects of the affection that I propose to deal.

Both carcinoma and sarcoma are liable to affect the larynx, but carcinoma is infinitely of greater frequency than sarcoma, and the variety attacking the larynx is mostly squamous epithelioma. My remarks, therefore, will apply to carcinoma larynx. Cancer of the larynx is met with most frequently between the ages of forty and seventy, and the decade fifty to sixty is the one in which cancer predominates. Males are undoubtedly more prone to the disease than females: Baratoux's statistics give two hundred and sixty-five males and only thirty-six females. Other authorities corroborate these statistics. No really satisfactory reason exists as an explanation of the greater liability of males to the disease.

In considering carcinoma of the larynx, it is of the greatest importance to adopt Krishaber's division of the disease into *intrinsic* and *extrinsic*; not only is this division of pathological importance, but it is invaluable from a prognostic and operative point of view.

By intrinsic cancer of the larynx is meant a tumor arising in connection with the true or false vocal cords,—the parts immediately below the true vocal cords and the ventricles.

By extrinsic cancer of the larynx is meant those tumors which grow from the epiglottis, aryepiglottic folds, the introarytenoid fold, etc.

Luckily, intrinsic carcinomas are much more frequent than

extrinsic' carcinomas. Baratoux collected one hundred and sixty-seven cases, of which one hundred and seventeen were intrinsic and fifty extrinsic. In Sendyiak's tables mention is made of one hundred and eighty-eight intrinsic and eighty-six extrinsic cases.

From my own personal observations and those of others in England, of the intrinsic parts attacked, the true vocal cords are most frequently the seat of disease; of the extrinsic parts, without doubt, the epiglottis.

The question of involvement of the neighboring glands is of the greatest interest and importance, and herein lies one of the most valuable reasons for Krishaber's division of laryngeal cancer into those of intrinsic and extrinsic origin. Some years ago Krishaber laid down as a general rule that the extrinsic cancers affect the glands early, whereas the intrinsic cancers, so long as they are limited to the cavity of the larynx, do not affect the glands. It is at once apparent that if this law be correct, it is one of the greatest importance not merely from a pathological point of view, but clinically. Although there are differences of opinion on the subject, those best able to judge of this rule of Krishaber's agree that it is in the *main* correct. Schwartz, out of seventeen cases of intrinsic cancer, observed glandular involvement in only four; and out of eleven cases of extrinsic, in nine cases.

The reason why intrinsic carcinomas of the larynx continue to grow for months and longer, even to a fatal termination, without glandular involvement, is at present not adequately accounted for. From work done by myself on this subject, I consider the general immunity of the glands in intrinsic carcinoma of the larynx to be due to the anatomical arrangement of the lymphatic vessels in the larynx, and the causation of non-glandular involvement to be of mechanical origin. The subject is, however, too extensive to deal with here.

The picture presented by an advanced carcinoma of the larynx is such that its diagnosis should, to any one skilled in laryngoscopy, be a matter of no difficulty. The age of the patient, the extensive and characteristic ulceration of the affected parts, their extensive destruction, the progress, slow usually, yet always progressive, the pain, particularly pain radiating to the ear, and later in extrinsic carcinomas the involvement of the cervical glands, present characteristics which exclude all doubt of the malignant nature of the

affection. But it is in the earlier stages of the disease that difficulties are likely to arise in the diagnosis, and it cannot be too much insisted on that only by an early and accurate diagnosis can any real benefit accrue to the patient by operative treatment.

The main points demanding especial attention in diagnosis of early malignant disease of the larynx are the age and sex of the patient, the existence of hoarseness (if intrinsic) without any discoverable cause, the presence or absence of any constitutional diathesis, the laryngoscopic appearances, and the examination microscopically of a fragment or fragments of the growth by a reliable pathologist. From *chronic laryngitis* early malignant disease can be diagnosed, as a rule, by its unilateral character and by the impaired mobility of the affected cord. This "lameness" is frequently observable at a very early stage, but varies, of course, according to the situation of the disease. It is, however, a symptom of great import.

From *innocent laryngeal tumors* (papillomata fibromata, etc.) the presence or absence of infiltration, the multiplicity of the growths, and the age of the patient constitute the main differential points. Moreover, the site of origin of the growth is particularly important to observe. Usually malignant growths spring from the middle or posterior third of the vocal cord: the presence of a growth in these positions in a patient over fifty years of age should always give rise to a suspicion of malignancy, for they certainly are not the usual sites of origin of benign papillomata. In all doubtful cases a piece of the growth should be removed by endolaryngeal forceps, and be submitted to microscopic examination. If, after removal of an apparently innocent growth, rapid recurrence ensues, malignant disease should be suspected; and it is well to bear in mind that in some cases, although microscopic investigation reveals no evidence of malignancy, yet the clinical appearances of the disease may be so typical of cancer that the surgeon has to rely more on the clinical than the microscopic evidences.

Pachydermia laryngis presents so typical an appearance that no difficulty should arise in the differential diagnosis from malignant disease. Besides, the absence of hoarseness, the history of alcoholism, and the free mobility of the cords are strong aids to a correct diagnosis of *pachydermia laryngis*.

From *syphilis*, the diagnosis may be replete with difficulties.

At present, a man of twenty-six is under my care with a hard nodular sessile growth occupying the posterior third of the left vocal cord, with marked infiltration of the interarytenoid space, hoarseness, difficulty in breathing, slight pain, and noticeable loss of mobility of the affected cord. The laryngoscopic appearance is such that in an older man a diagnosis of malignant disease would probably be made, yet the whole of my patient's trouble is due to syphilis. He was under my care some five or six years ago for well-marked secondaries affecting both the throat and skin, and rapidly improved under mercury. Yet in this particular patient careful examination of the larynx reveals one or two points differing from malignant disease; the marked infiltration of the interarytenoid space is cicatricial in nature; this cicatrization is quite common as a result of gummatous affections of the larynx, and often leads to severe stenosis. Never in carcinoma do you find cicatrization. Again, there has been a marked absence of pain until a few days before seeing me. This absence of pain is usual in syphilitic ulceration of the larynx, and contrasts with the great pain almost invariably present in malignant ulceration.

In cases of doubt, other manifestations of syphilis may be present; and at all events, in such cases, the patient should at once be put on large doses of iodide of potassium and careful note made of any changes in the size or extent of the ulcers or infiltration.

From *tubercle*, the diagnosis usually presents no difficulties. The younger age of the patient, the bilateral character of the disease, the multiplicity and curious "mouse-nibbled" appearance of the ulcers, the swelling and pallor of the surrounding parts, the condition of the lungs, and presence of the tubercle bacillus in the sputa form a clinical picture quite different from that of malignant disease. Cases do, however, arise in which a correct diagnosis presents difficulties. Some months ago I had under my care a patient in whom I diagnosed carcinoma laryngis. Two of my colleagues agreed with my diagnosis; and another, a skilled laryngologist and diagnostician, was sure the case was one of laryngeal tubercle. Subsequent events proved the nature of the disease to be malignant.

The treatment of malignant disease of the larynx consists (1) of radical and (2) of palliative treatment. *Radical treatment* is further divisible into:

(a) Endolaryngeal removal.

- (b) Suprathyroid laryngotomy.
- (c) Infrathyroid laryngotomy.
- (d) Thyrotomy or laryngo-fissure.
- (e) Partial resection of the larynx.
- (f) Total removal of the larynx.

Of removal of malignant disease of the larynx by endolaryngeal methods, I may say at once that it is only under very rare conditions that it should be practised. If the disease be very limited in extent, the patient very old, and the general health bad, or the patient refuses the major operation, an attempt may be made to extirpate the disease *per vias naturales*. Except under such circumstances, I strongly condemn endolaryngeal removal of cancer of the larynx. It is practically impossible to make certain of complete removal of the disease; the operation generally has to be repeated, and a possibly sluggish growth stimulated into a more active one, and recurrence is almost invariably the rule. I know of no authentic cure of malignant disease of the larynx by this method.

Suprathyroid laryngotomy has been advocated in cases where the disease is limited and confined to the upper opening of the larynx, especially the epiglottis, and *suprathyroid laryngotomy* for removal of growths on the under aspect of the cords or actually below the cords. In both conditions a thyrotomy will equally meet the case, and is preferable on account of the greater space available to deal with the disease. It is the common experience of those who have had to operate for laryngeal cancer often to find the disease extending over a larger area than had been considered to be the case from the appearances as seen by the laryngoscope, and under such circumstances thyrotomy is infinitely a more satisfactory operation.

Thyrotomy or *laryngo-fissure* is *par excellence* the operation for laryngeal cancer, particularly so if of intrinsic origin. It has come into general use and favor in England mostly from the excellent results obtained by it by Butlin and Semon, both of whom are mainly responsible for its introduction into this country, and for very valuable modifications in the after-treatment, rendering the operation one far less fatal than it used to be. The best results are obtained from it in those cases of intrinsic carcinoma in which the disease is limited to the interior of the larynx. It is also suitable, as I have before said, for cases in which the disease is limited

to the epiglottis, or is situated on the under surface of the vocal cords or just below them. A moderate involvement of the cervical glands is no contraindication to the operation, as they can be removed by a separate operation some two or three weeks later. The value of Krishaber's division into carcinomas of intrinsic and extrinsic origin again strikes one in discussing thyrotomy. Intrinsic carcinoma is in the early stages nearly always of limited malignancy, with but little, if any, tendency to glandular infection, and no tendency to dissemination. It attacks the cartilaginous framework of the larynx with difficulty, so that, even when the surface of the cartilage is attacked, only the face of the cartilage, as a rule, requires removal.

The operation of thyrotomy, for removal of laryngeal cancer, as practised in England, is briefly as follows. An incision is made from the hyoid bone in the midline of the neck down almost to the sternum, and the structures divided right down to the thyroid cartilage and trachea. The trachea is opened below the cricoid cartilage and a Hahn's sponge cannula inserted. After an interval of ten or fifteen minutes, to permit the expansion of the compressed sponge, the thyroid cartilage which has been exposed and cleared by the preliminary incision is carefully split in the midline from below upward. Often, as the patients are old people, the cartilage is ossified, and a fine saw may be required for its division. The cricothyroid membrane is divided downward and the incision carried upward well above the upper border of the thyroid cartilage to give as much room as possible. The two alæ of the thyroid cartilage are held apart, the interior of the larynx sponged dry, and a solution of adrenalin and cocaine applied to lessen hemorrhage and the sensibility of the parts. Two elliptical incisions are now made down to the perichondrium so as to include the diseased tissues and a good half inch of the surrounding apparently healthy tissues. The included area is excised and the exposed cartilage scraped. After checking any hemorrhage, which is usually slight, the two alæ of the thyroid cartilage are accurately brought together with silk or silver sutures, the Hahn's tube removed, and the edges of the incision in the soft parts brought together and sutured, except at the lower part. It is important to note that the Hahn's tube is removed at once, and no tube inserted into the trachea. This procedure was introduced by Butlin, and has given uniformly good results. It

removes a very possible source of septic infection, for in some of the fatal cases recorded death ensued from sepsis, the origin of which was undoubtedly the compressed sponge of the cannula, which had been left in for twenty-four to forty-eight hours. Butlin has also abolished plugging the interior of the larynx with strips of iodoform gauze, as he found that they acted as irritants, and occasionally got dislodged, hanging down into the trachea and acting as foreign bodies. The only dressing necessary is a piece of cyanide gauze over the wound, the gauze to be changed as often as is necessary. The patient is kept on his side, that side being lowermost which corresponds to the half of the larynx operated on, and the head should be kept low. In this position all fluids tend to pass out of the air passages through the external wound. The day of the operation nothing is swallowed, except, possibly, some small pieces of ice. The morning following the operation an attempt is made to give fluids by the mouth. The patient is propped up and made to lean well forward, and to take cold water out of a glass. If successful, the water passes into the stomach; if unsuccessful, it escapes through the larynx, the position of the patient, however, preventing its passing down into the air passages. As soon as water can be easily swallowed, milk, beef-tea, etc., may be given. If all goes well, the wound is closed by the tenth to the twelfth day, and the patient is convalescent within a fortnight. The operation as I have described it applies only to those cases in which the disease is of small extent and limited to the soft parts. In many cases the operation turns out to be more extensive, even to the extent of removal of a large part, or even the whole, of the cartilaginous framework.

Partial and total extirpation of the larynx have been practised abroad much more frequently than in England. English laryngologists are not, on the whole, in favor of total laryngectomy, although a fair number of partial laryngectomies has been performed. Both operations, especially total laryngectomy, have a high rate of mortality, and the ultimate results, when not fatal, have been far from good. I would advise partial laryngectomy in cases in which, while too extensive to deal with by thyrotomy, there were justifiable reasons to suppose that complete removal of the disease could be accomplished by its performance, and in which the patient was not too advanced in age or the general health too bad; also in cases in which rapid recurrence had followed a previous thyrotomy. Of

total laryngectomy I can say but little that is favorable. If the disease be too extensive to be adequately dealt with by excision of half the larynx, it appears to me very doubtful that it could be completely removed by even total extirpation of the larynx. The mortality is very high, particularly from pneumonia and bronchitis; and if the patient survives the operation, recurrence is the rule, and in all cases the patient's subsequent condition is a far from enviable one.

Palliative Treatment.—Tracheotomy can with confidence be advised in those cases of laryngeal cancer considered to be inoperable, and in which there is increasing dyspnoea. Vauvel states that in seven patients suffering from cancer of the larynx on whom tracheotomy was performed, the average duration of life was four years; whereas in six patients suffering in the same way, and in whom no operation was performed, the average duration of life was twenty-one months.

Pain must be met by suitable insufflations of morphine or orthoform, and ulceration and fetor by inhalations of creosote or insufflation of iodoform, cocaine, and hydrochlorate of morphine.

In conclusion, let me emphasize the necessity for an early diagnosis of laryngeal cancer; if intrinsic, thyrotomy is the operation best suited to deal with the case, and should be advised without delay.

If extrinsic, the prognosis is much less favorable for operation; there is greater local malignancy, infection of the neighboring glands is apt to occur early, the actual removal of the disease is more dangerous to life, yet, if still limited to the interior of the larynx and the disease be of small extent, very good results also follow an early thyrotomy.

TREATMENT OF ANEURISMS BY GELATIN IN HYPODERMIC INJECTIONS.

BY E. LANCEREAUX, M.D.,

Physician to the Paris Hospitals.

SINCE my first paper on this subject was published in June, 1897, the method has been employed in almost every part of the world; not only has it been used for aneurisms, but it has been tried in different kinds of hemorrhage, such as hæmoptysis and purpura, and in the hemorrhagic forms of the eruptive fevers. In the majority of cases of aneurism thus treated the results obtained have been satisfactory, since the aneurismal pouch has almost always hardened, the movements of expansion have disappeared, and with them the painful symptoms. On the other hand, a certain number of failures have been reported; but, when the cases are closely examined, it is found that these failures were due to two great causes: (1) The treatment was applied to cases in which it could not possibly have any effect, as where there was fusiform ectasis of the artery. (2) The *modus operandi* was arbitrarily modified: either the doses of the gelatinous solution used were inadequate (from twenty to thirty cubic centimetres instead of from two hundred to two hundred and fifty) or the number of injections given was too small (two or three, instead of fifteen, twenty, or thirty).

I have already laid stress on the uselessness of injections of gelatin in cases of simple ectasis of the artery, where there is no aneurismal pouch properly so called; and I have shown that under such conditions coagulation does not take place, because the flow of blood is not sufficiently slackened, and that the latter factor is one of the indispensable conditions to the formation of clots in the interior of a vessel.

I have at the same time endeavored to ascertain the amount of gelatin necessary to increase to a slight degree the power of coagulation of the blood, and I have reached the conclusion by calculation, and by clinical experimentation and observation, that the minimum dose of gelatin, which will be easily and readily absorbed when it is

dissolved in from two hundred to two hundred and fifty cubic centimetres of a seven-per-thousand solution of chloride of sodium, ought to be about five grammes. It is, therefore, not astonishing that doses of twenty or thirty cubic centimetres of this solution have proved to be inefficacious. I have found that after such an injection of gelatin the contents of the aneurism coagulate, the tumor hardens very quickly, the expansive movements diminish, and the pain decreases; but in a few days both movements and pain reappear. If, then, another injection is made, it produces the same results as the first one, and, if the series is continued up to ten, fifteen, thirty, and even thirty-five injections, definite recovery can be effected.

It seems to me that these results can be interpreted somewhat in the following way. The clot that is formed at the first injection obturates the greater portion of the aneurismal pouch; but, since it soon undergoes retraction, it allows some blood to enter the pouch again. This fresh blood is coagulated by the next injection, and the process thus goes on until a sufficient number of injections have been made to fill the pouch entirely with strong and resistant clots. It will be readily seen that the number of injections necessary to obtain this result will depend on a variety of circumstances, and particularly on the size of the aneurism. For this reason it is easy to understand that two or three injections will procure only temporary relief for the patient, and not a definite cure of the aneurism.

In reply to the criticism that the injections are painful and cause a rise of temperature, I can only say that when the gelatin solution is slowly injected into the cellular tissue of the hip it causes no pain; and, on the other hand, if careful aseptic precautions be taken the procedure is not followed by a rise of temperature.

I think it necessary to dwell on these details to show that the failures with my method must be attributed rather to the defective way in which it was applied.

I wish now to refer to two new patients, one of them suffering from an aneurism of the right subclavian artery and the other from a similar disorder of the ascending aorta, both of whom have been considerably improved, if not cured, by hypodermic injections of a solution of gelatin. The former of these patients had an enormous aneurismal pouch of the subclavian artery, whose contents consisted clearly of liquid blood, since the tumor was soft and reducible. At the present time it is easy to see on examination that, under the in-

fluence of injections of the gelatinized serum, the pouch has become as hard as wood, and no more expansive movements can be detected in it. In the second of these patients, a woman suffering from an aneurism of the ascending aorta that pressed on the upper vena cava and on several intercostal nerves, the subcutaneous injections of gelatin put an end altogether to the neuralgic pain, and lessened considerably the collateral venous circulation that was taking place in the anterior portion of the thorax.

I have also this year taken care of two other similar patients, of whom I may say a few words. The first of them, a syphilitic woman fifty years of age, showed a voluminous aneurism of the aorta pressing on the upper vena cava (collateral venous circulation of the upper portion of the thorax), on the intercostal nerves (neuralgic pain), and on the pneumogastric and recurrent nerves (incessant spells of hoarse, barking cough). After six injections of gelatin, first the cough and then the pain disappeared entirely and the collateral venous circulation decreased. Thinking herself cured, she left the hospital, but was seized again not long afterwards with pain and fits of coughing. She then returned to our wards, where she is again undergoing the gelatin treatment and her condition has rapidly improved. The other patient, a woman of sixty, suffering from arteriosclerosis and an enormous aneurism of the aorta, which had destroyed the top of the sternum and a part of the collar-bone and first rib, has been considerably improved, if not cured, after a series of twenty injections of gelatin; this woman, when she left the hospital, went back to her work, and I saw her some time afterwards, and was able to satisfy myself that the improvement in her condition had been maintained.

Consequently, I am of opinion that at the present time these injections of gelatinized serum constitute the only method of treatment that is harmless and capable of curing true aneurisms of the aorta that are not applicable to surgical treatment.

SELECTED PRESCRIPTIONS.

COUGH MIXTURES.

For a sedative cough mixture in the productive stage of an acute bronchitis, Scott's old-time prescription has of late dropped somewhat out of sight. It deserves to be remembered, especially when the cough has become racking and the muscles are sore from the unaccustomed strain put upon them.

R Ammon. carbonat., ℥ii;
Tinct. bellad., f℥i;
Tinct. opii camphorat., f℥ss;
Syr. scillæ, f℥v;
Syr. senegæ, q.s. ad f℥iii.—M.

Sig.—One teaspoonful four or five times a day.

For slight coughs in delicate people, especially in the old, it must not be forgotten that the aromatic spirit of ammonia makes an excellent stimulating expectorant. There are no depressing effects from its use which many cough remedies produce because of their ingredients, nor is it liable to upset a capricious stomach, or lessen an already unsatisfactory appetite. It may be given with excellent effect in such cases in combination with cherry laurel water, which is much more popular in Germany than in this country.

R Spt. ammon. aromat., f℥iss;
Aq. laureæ cerasi., q.s. ad f℥iii.—M.
Sig.—Teaspoonful every two hours.

For children, its use in combination with a syrup, is often advisable.

R Spt. ammon. aromat., f℥i;
Syr. pruni virginian., q.s. ad f℥iii.
Sig.—Teaspoonful every two or three hours.

For chronic cough in old patients, where cough medicine must be used over long periods, Child's ammonia mixture is very valuable and deserves its original popularity. It has been somewhat lost from sight in the midst of the many proprietary cough remedies forced upon the notice of the physician in recent years.

R Liq. ammonii anisat., f℥i;
Spt. frumenti,
Syr. simp., ℥ss;
Aq. q.s. ad f℥iii.—M.
Sig.—Teaspoonful every two or three hours.

In robust patients, for the severe cough of a very acute bronchitis, the value of aconite in quieting the circulation, reducing the temperature and so lessening the irritative tendency to cough, should not be forgotten. Coughs that have not yielded to any of the expectorants even when given in large doses, promptly succumb to aconite. It may be prescribed very effectively in the following mixture:

R Tinct. aconiti, $\mathfrak{m}_{\text{xvi}}$;
 Ammon. carbonat., gr. xl;
 Tinct. opii camphorat., \mathfrak{f}_{3v} ;
 Syr. pruni virginianæ, q.s. ad $\mathfrak{f}_{\text{3ii}}$.—M.

Sig.—Teaspoonful every two hours until the cough softens.

FLAXSEED TEA FOR COLDS.

This beverage, if drunk from time to time, has a distinctly emollient effect in severe colds. For delicate children it is especially indicated. It possesses no tendency to upset the stomach that so many of the syrup cough medicines do, and, besides, is distinctly nutritious, comparing favorably with barley or rice syrup in this respect. Flavored with a little vanilla or mixed with a tasty proportion of lemon juice, it is very savory, and children seldom refuse to take it. The cough of an acute bronchitis is usually accompanied by a daily rise of temperature. This not only disturbs the appetite, making it important that nutritives should be advisedly employed, but also produces a tendency to constipation. Flaxseed tea is one of the commonest remedies in Germany for continued constipation, and can usually be depended upon to produce a gentle laxative effect if given in ounce doses about three times a day. Recently, the presence of the seeds has been urged against the remedy even by so good an authority as Boas because of the danger of their causing appendicitis. The objection seems far-fetched, however, since, notwithstanding the frequency with which flaxseed tea has been used, no case of appendicitis from the seeds has yet been reported. Flaxseed tea is made by boiling a heaping tablespoonful of the unground seed in a quart of water for two hours. The decoction should then be strained through a linen cloth.

CHRONIC BRONCHITIS.

For the cough of chronic bronchitis, especially when the patient is suffering from an acute exacerbation of the symptoms, the classic formula is:

R Potassii iodidi, ℥iii;
 Tinct. bellad., f℥ii;
 Spir. ætheris comp., f℥i;
 Ext. pruni virgin. fl., f℥iiss;
 Syr., f℥i;
 Aq. dest., q.s. ad f℥iv.—M.

Sig.—Teaspoonful four or five times a day.

When the stomach is easily upset, equal parts of aromatic spirit of ammonia and peppermint water may be substituted for the syrup with good results.

ASTHMA.

For asthma due to bronchial spasm without chronic inflammation of the mucous membrane of the bronchi, the following prescription is highly recommended:

R Potassii iodidi, ℥v;
 Tinct. lobeliæ, f℥x;
 Spt. glanoini (1 per cent.), gttæ xv;
 Elix. potas. bromid. (N. F.), ℥iv.—M.

Sig.—One teaspoonful three times a day after meals.

If the asthmatic attacks occur during sleep, a double dose may be taken just before going to bed. The dose should be gradually increased if necessary to double doses three times a day.

BRONCHIECTASIS.

The following mixture has been recommended in this affection because it makes the secretion in the enlarged bronchi less fetid, more fluid, and more easily expectorated.

R Eucalyptol, mxx;
 Creosoti, ml;
 Tinct. benzoini, f℥iiss;
 Copaibæ, ℥iiss;
 Ol. amygd. dulc., ℥vi.—M.

Sig.—Inject twenty to thirty drops in a small amount of milk into the rectum in the early morning. The dose should be gradually increased until one or two teaspoonfuls are employed. The treatment should be kept up for some months. Its efficiency can then be fully appreciated.

When the expectoration of bronchiectasis is copious and the patient has some difficulty in emptying the bronchial dilatations of their contents, he should be directed to lean out of bed in the morning with his hands on the floor. Gravity thus aids in the evacu-

ation of the bronchiectatic collections of fluid. For the very offensive odor that sometimes makes life miserable for the patient and those near him, two teaspoonfuls of the syrup of garlic (*Syrup. alii*) taken morning and afternoon, or oftener, are said to be effective. The odor was removed by the puncture of the principal cavity by means of a Paquelin cautery in at least one reported case. Crede's ointment is recommended for the prevention of the metastatic abscesses that are sometimes noted and that occur occasionally in the brain, remaining latent often for long periods.

For milder cases, the offensive odor of the secretions and the spasmodic cough which these secretions are apt to occasion, may be overcome by the inhalation of the following:

R Formalin, ℥ii;
Ac. carbolic, ℥i;
Tinct. opii camphorat., q.s. ad ℥iv.—M.

Sig. —One teaspoonful to one-half pint of hot water, to be freely inhaled morning and afternoon.

DIARRHŒA FROM COLD.

For the diarrhœa that follows exposure to cold after a hearty meal, or that comes on from throwing off the bedclothes at night, the following is recommended:

R Tinct. capsici,
Tinct. opii,
Spt. camphoræ, aa f ℥iiss;
Chloroformi, f ℥;
Alcoholis, q.s. ad f ℥ii.—M.

Sig.—One teaspoonful every hour until relieved of pain; then one teaspoonful every two hours until the diarrhœa is effectually checked.

FOR CHRONIC RHEUMATISM.

For genuine chronic rheumatism, that is a condition of stiff and painful joints that has developed as the result of a number of attacks of subacute rheumatism, the following formula has been in vogue for many years:

R Sodii bicarb.,
Potassii acetat., aa ℥vi;
Sodii et potassii tartrat., ℥iiss;
Spt. chloroformi, f ℥i;
Aq., q.s. ad f ℥vi.—M.

Sig. —One tablespoonful three or four times a day two hours before or after meals.

THE TRIPLE BROMIDES.

A favorite formula for those who consider the combination of three bromides more effective and less depressing than any single bromide is the following:

R Potassii bromidi, ℥ss;
Sodii bromid.,
Ammonii bromid., aa ℥ii;
Elix. aromat., ℥iii;
Aq. q.s. ad f ℥vi.—M.

Sig.—One tablespoonful three times a day.

An even pleasanter prescription can be made by combining the elixirs of the bromides of the national formulary in equal parts, and there is then no need to have recourse to proprietary bromide preparations.

UTERINE SEDATIVE.

There are so many proprietary remedies constantly being pushed into prominence that are supposed to respond to such indications as dysmenorrhœa or metrorrhagia, that a reliable prescription, the ingredients of which can be varied so as to suit particular cases, should be especially welcome.

R Ext. viburni prunifol. fld.,
Ext. ergotæ fld.,
Glycerite hydrastis,
Syr. aurantii, aa f ℥i.—M.

Sig.—One to two teaspoonfuls three or four times a day.

HEADACHE POWDERS.

The coal-tar products are extensively and very successfully used for headache. There is no doubt that harm is being done, however, by the excessive administration of these powerful remedies. There is always a nervous condition associated with a headache that has continued for any length of time that makes even slight pain unbearable to sensitive people. For this neurotic condition the coal-tar products are of no avail, and so much larger doses must be employed to get entire relief from the headache. The bromides are, however, of excellent service for the sedation of the associated general nervous condition, and, practically, should be always combined with the coal-tar product employed. The following combination has been found especially valuable for the reasons stated.

R Potassii bromidi, $\mathfrak{Z}\text{i}$;
 Caffeinæ citratis, gr. iii;
 Phenacetine, $\mathfrak{Z}\text{ss}$;
 Sodii bicarbonatis, $\mathfrak{Z}\text{ss}$.

M. et divid. in chart. No. vi.

Sig.—One powder to be repeated every half-hour until three are taken, if necessary.

Some prefer to prescribe acetanilid instead of phenacetine, and then the amount should be only one-half that suggested in the prescription.

RHEUMATIC DISCOMFORT IN THE OLD.

For the pains and stiffness in the joints that develop in old persons and are more noticeable in damp weather, a combination of iodides and some colchicum preparation is the most effectual remedy. When the condition is not true rheumatism, there is usually no history of acute or subacute rheumatic arthritis, and the salicylates fail, as a rule, to relieve the symptoms. The following formula is used in several of the out-patient departments of New York City Hospitals:

R Potassii iodidi, $\mathfrak{Z}\text{iii}$;
 Vini colchici radicia, $\mathfrak{Z}\text{liiss}$;
 Syr. sarsap. comp., f $\mathfrak{Z}\text{ii}$;
 Aq. q.s. ad $\mathfrak{Z}\text{iv}$.—M.

Sig.—Two teaspoonfuls after meals.

TONIC PILL.

An excellent tonic pill for those who find it difficult to take a bitter tonic in liquid form, or for those whose occupations make it difficult for them to have a bottle with them at the time they wish to take their medicine, is the following:

R Quin. sulphat.,
 Fe. sulphat. exsicc., \mathfrak{aa} gr. xii;
 Ext. nuc. vomicæ, gr. iv;
 Acidi arseniosi, gr. $\frac{1}{2}$.—M.

M. et ft. pil. No. xii.

Sig.—One pill before meals three times a day.

WHOOPIING COUGH.

Notwithstanding the many remedies introduced for this obstinate affection, belladonna and antipyrin are still generally acknowledged to be the most effective in reducing the number and severity of the coughing spasms.

R Tinct. belladonnæ, ℥i;
 Antipyrini., gr. i;
 Sodii bromidi, ℥ii;
 Syr. pruni virginian., f ℥i;
 Aq. q.s. ad f ℥iii.—M.

Sig.—One teaspoonful four or five times a day.

For those who dread the use of antipyrin, even in small doses, phenacetine may be substituted, but, because of its limited solubility, should be prescribed separately in two or three grain doses.

EXCESSIVE SWEATING OF THE FEET.

This affection may become a source of very great annoyance if not taken in time. The Journal of the American Medical Association recently suggested two prescriptions for it, the first to be used especially after the evening bath, the second to be dusted over the feet before the stockings are put on in the morning.

R Balsami peruv., ℥xv;
 Acid. formici., ℥i;
 Chloralis hydrat., ℥i;
 Alcoholis, q.s. ad f ℥iii.—M.

Sig.—Apply on a swab of absorbent cotton after the evening bath.

R Olei lavandulæ, ℥xii;
 Olei caryophylli. ℥iv;
 Tannoformi, ℥i;
 Acid. borici, ℥iv.—M.

Sig.—Use freely as dusting powder in the morning.

NUTRITIVE LEMONADE.

There is need of greater variety than is at present usually suggested in the matter of liquid diet. Patients grow tired of milk, beef tea and barley water. The recipe for making a white-of-egg lemonade that should make a valuable addition to the liquid dietary is as follows:

Take two lemons, the yellow rinds of which are used for flavoring, the inner white rinds are thrown away. Slice the inside of the lemons. Pour over the slices and yellow rinds of the lemons a pint of hot water. Stir until cooled to the temperature of ordinary tea, and then strain. The whites of two eggs are then slowly added, the liquid meanwhile being briskly stirred. The mixture is whipped for several minutes and then strained. Sugar to taste. Serve cold.

The albumen gives a blandness to the drink that makes much

sweetening unnecessary, and this absence of sweetish taste makes the drink especially agreeable to fever patients. Needless to say, the beverage, while apparently only a simple thin liquid, is very nutritious because of its egg and sugar contents. It is especially indicated in typhoid fever, but contraindicated in Bright's disease and gastric ulcer. In the former because it increases the albuminuria, in the latter because it heightens the already excessive acidity of the stomach.

ANOREXIA.

For loss of appetite due to mental overwork or physical lassitude, the following prescription has long been a popular favorite, especially in England.

R Tinct. nuc. vomic. ;
Acid. hydrochlorici dil. ;
Spt. chloroformi, aa f ℥iij ;
Aq. menth. piperit., q.s. ad f ℥iij.

This is especially useful in the anorexia due to over-indulgence in alcohol also, and it is without the disadvantage of so many tonics which supply an undue amount of alcohol to the patient in the shape of tinctures of bitter principles.

Medicine

REPORT OF A CASE OF PERNICIOUS ANÆMIA WITH EXTENSIVE PIGMENTARY CHANGES IN THE SKIN.

BY THE LATE FREDERICK A. PACKARD, A.M., M.D.,
Of Philadelphia.

THE subject of this report was a white male aged thirty-seven years, an American by birth, and a conductor by occupation. He was first admitted to the Pennsylvania Hospital, under the care of my colleague, Dr. A. V. Meigs, on January 19, 1901.

The family history is unimportant.

In his past history there is nothing bearing on his case except an attack of pneumonia at the age of eight years and one of rheumatism at twenty-one years. In the latter part of June, 1900, he had an attack of what was said by his physician to be inflammatory rheumatism; the main symptoms were pain in the left side and vomiting. After an illness of three weeks his acute symptoms subsided, but since that time his health has steadily failed. He progressively lost weight, from one hundred and sixty-eight to one hundred and thirty-one pounds, and became steadily weaker and paler. His chief complaint, aside from weakness, was of severe pain in the upper abdomen, in the back, and in the legs.

On admission he was found to be very pale, his skin being of a lemon-yellow color and his mucous membranes of a dull white. He was much emaciated. His tongue was flabby, slightly coated, and fissured. The pulse was fairly regular, but was very compressible. He had a temperature of 101° F., pulse 96, respirations 24. The chest was covered with curious alternations of pale and pigmented skin, described more fully below.

Examination of the heart and lungs showed no abnormality. There was no change in the size either of the liver or of the spleen. The abdomen was scaphoid, slightly tender in the region of the gall-

bladder, and with a sense of resistance to palpation in the median line between the navel and the xiphoid; but no distinct mass could be felt. The urine was amber-colored, of acid reaction, and its specific gravity 1020; it contained neither albumin, sugar, nor casts.

The blood was examined on January 22, with the following result: erythrocytes 1,480,000, leucocytes 2080, hæmoglobin, 25 per cent. There were present microcytes, macrocytes, and poikilocytes. No nucleated red cells were found, but there was some polychromatophilia.

Polymorphonuclears	80 per cent.
Small lymphocytes	10 per cent.
Large lymphocytes	5 per cent.
Eosinophiles	5 per cent.

The man's general condition seemed to improve under Fowler's solution. On omitting this drug for a few days he appeared rapidly to lose ground, and at the end of a week arsenic was again started. On February 17 the erythrocytes were 1,092,000, leucocytes 5133, hæmoglobin 25 per cent.

On March 8 the erythrocytes were 1,272,000, leucocytes 5266, hæmoglobin 22.5 per cent.

Polymorphonuclears	79.67 per cent.
Small lymphocytes	14 per cent.
Large lymphocytes	0.4 per cent.
Myelocytes	0.8 per cent.
Eosinophiles	5.2 per cent.

There were numerous myelocytes; no nucleated red cells but many polychromatophilic degenerated corpuscles were found.

In spite of the continued poverty of the blood, the man had gained seventeen pounds after residing two and a half months in the hospital. On systematic routine examination made on April 7 he was described as a fairly well-nourished man, with skin of pale yellow hue, with irregularly distributed areas of leucoderma about the eyes and front of the neck and chin. The mucous membranes were pale and showed no pigmentation; the tongue was of a pale pink and smooth. The large portion of the trunk was very white, and scattered over it irregularly there were areas of brownish pigmentation with curiously scalloped edges. There was no change in the area of cardiac dulness, but at the apex there was a very

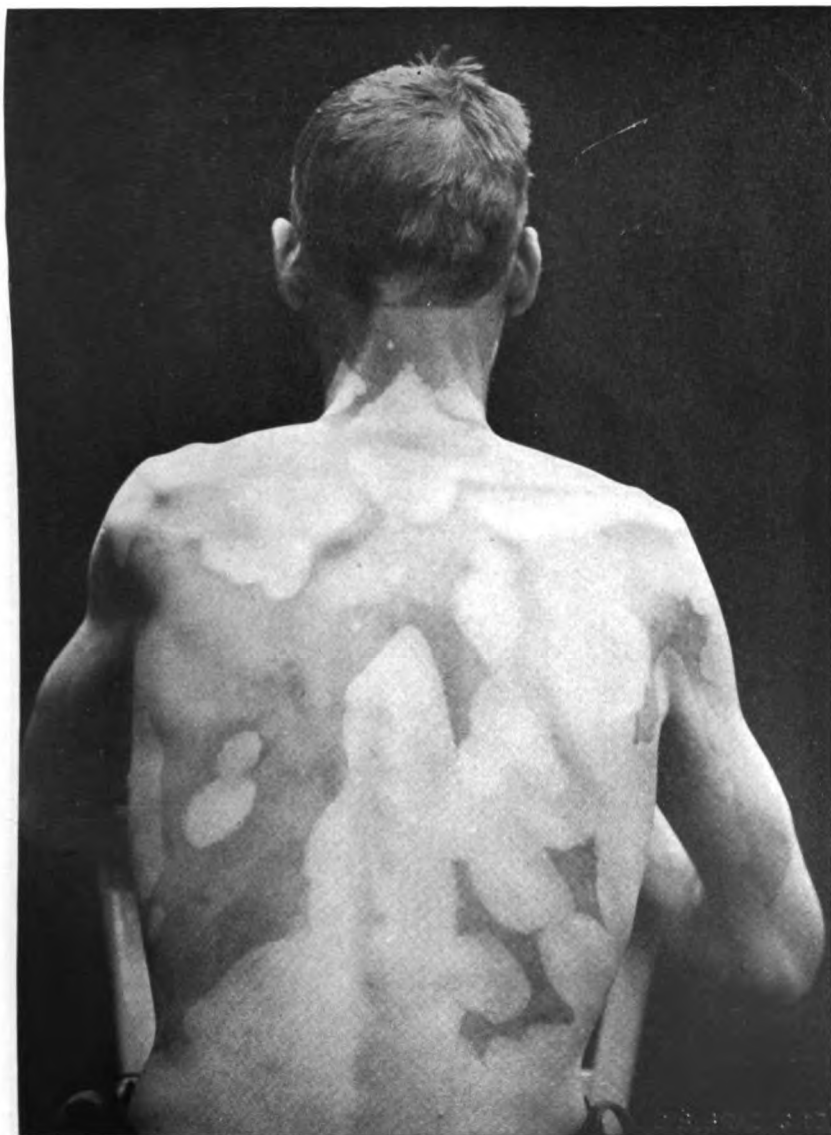


FIG. 1.—Pernicious anemia with marked pigmentary changes in the skin.

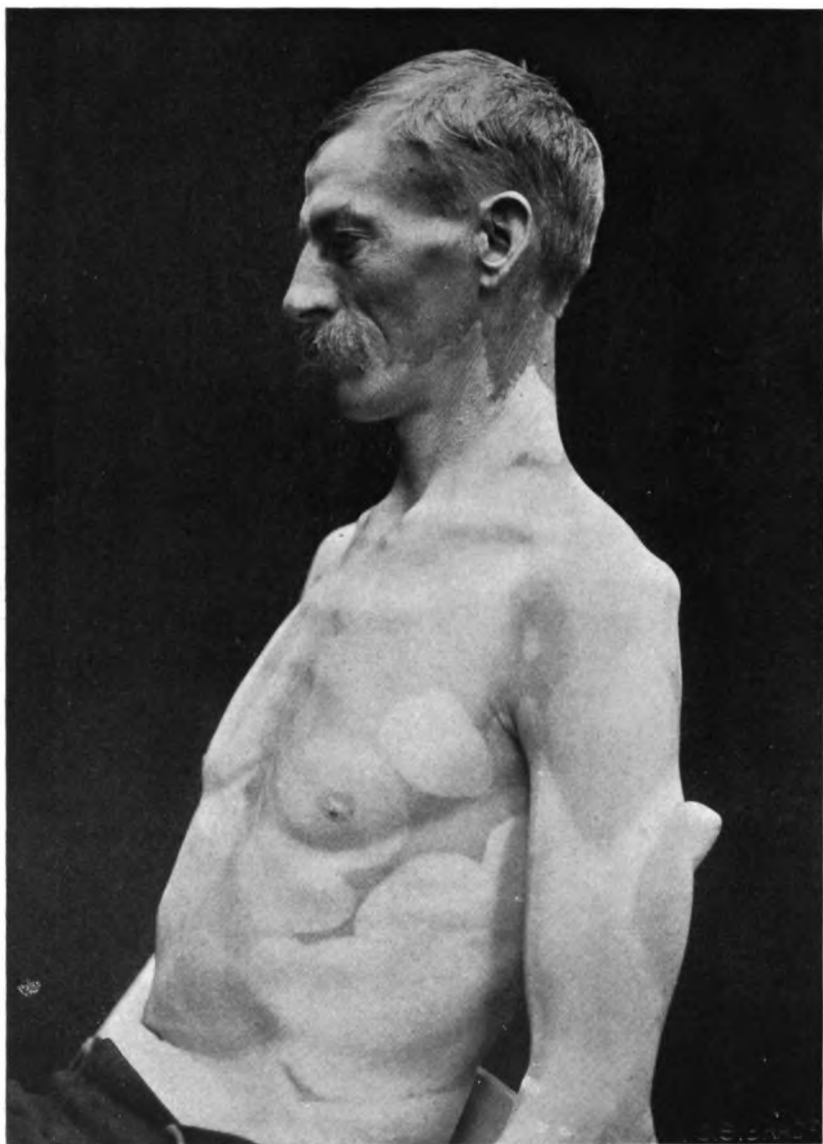


FIG. 2.—Side view of the same patient.

faint, soft, systolic murmur, not transmitted. At the pulmonary area there was a soft, systolic, blowing murmur. There was no venous hum in the neck and no lymphatic enlargement. The lungs showed no abnormality. The area of hepatic dulness extended from the lower border of the fifth rib to the upper portion of the seventh rib. The spleen was not enlarged. Over the general surface of the back the skin showed large white areas separated by areas of deep-brownish pigmentation, the dividing line between the pigmented and non-pigmented areas having a generally scalloped shape. Between the shoulder-blades there was a square area of deeper pigmentation where a blister had been applied early in the preceding June. (Figs. 1 and 2.)

Ophthalmoscopic examination showed that the eye-grounds were very pale and that the vessels were small. The optic disks were slightly smaller than normal. There were no signs of retinal hemorrhage, past or present.

The blood examination made at about this date showed erythrocytes 1,045,000, leucocytes 3500, hæmoglobin 21 per cent.

The patient's condition improved as regards his strength and weight, and at his request he was allowed to go home.

He returned to the hospital on May 2, and a few days afterwards examination of the blood showed erythrocytes 1,145,000, leucocytes 2480, hæmoglobin 15 per cent.

Analysis of the gastric contents after a test meal gave an alkaline reaction with no evidence of hydrochloric acid, lactic acid, pepsin, or milk-curdling ferment.

Examination of the urine at about this time showed a specific gravity of 1012, amber color, traces of albumin, uric acid crystals, no casts. Chemical and spectroscopic tests showed the presence of "pathological urobilin."

On May 23 nucleated red cells were found, and a differential count of the leucocytes showed—

Polymorphonuclears	60.9 per cent.
Small lymphocytes	33.6 per cent.
Large lymphocytes	2.4 per cent.
Eosinophiles	2.7 per cent.
Myelocytes3 per cent.

The patient insisted that the light skin was abnormal and that the lighter areas were increasing in size; to this statement he

adhered on repeated and close questioning. In favor of the view that the condition was one of vitiligo rather than chloasma was the almost universal circular shape of the margins of the areas of lesser pigmentation. The concave edges of these areas were such as would be made by lesions advancing at the periphery. Against the view that it was a case of vitiligo was the square area in the spinal gutter (well shown in the photograph) where deep pigmentation had followed the application of an irritant. The latter, however, would simply point to a trophic anomaly of the pigmentation of the skin in general, and can hardly be considered to outweigh the patient's statement and the shape of the lesions.

Decastello¹ has reported three cases of pigmentary changes in the course of pernicious anæmia. His first patient showed areas of pigment change corresponding to the metameric sensory zone described by Head. While no exact observations upon this point were made by me on the case reported, the distribution as seen in the photographs rather suggests an approach to the same correspondence. One of Decastello's patients presented the association of pernicious anæmia, Graves's disease, and vitiligo. Similar cases of pigmentary anomalies have been reported by Broadbent and Immerman.

The characteristic tint of the skin, enabling one almost to make the diagnosis of pernicious anæmia by inspection, has never been fully explained. Possibly the trophic changes in the pigmentation of the skin observed in the cases mentioned above and in that forming the subject of this report may be due simply to extraordinary development of the cutaneous changes ordinarily seen in pernicious anæmia, and the association may not be merely accidental.

Dr. Frank Billings, of Chicago, at the last meeting of the Association of American Physicians showed photographs of a patient with splenic anæmia and pigmentary changes almost precisely like those illustrated in the accompanying photographs.

Dr. William Osler has told me of two "remarkable cases of piebald pigmentation in pernicious anæmia," one of which had been diagnosed as Addison's disease, and a third case of diffused pigmentation in the same general disease.

¹ Wiener klinische Wochenschrift, December 26, 1901.

EXHIBITION OF LUNGS AND HEART FROM A CASE OF LEFT-SIDED PNEUMONIA WITH PURULENT PERICARDIAL EFFUSION.

BY FREDERICK P. HENRY, A.M., M.D.,

Professor of the Principles and Practice of Medicine, and of Clinical Medicine,
in the Woman's Medical College of Pennsylvania; Physician
to the Philadelphia Hospital.

GENTLEMEN,—The greatest advantage of hospital service, in my opinion, is the frequent opportunity it affords for observing the correlation between the physical signs of disease and the structural changes on which they depend. The study of a fatal case of disease is completed only by the autopsy, and, conversely, a post-mortem examination without a record of the clinical history is comparatively worthless. These remarks are suggested by the fact that the specimens which I now exhibit are those of a patient whom you have never seen. Your interest in them would doubtless be greater if, like me, you had had the opportunity of studying the case from which they were derived,—in other words, of determining for yourselves the correlation between them and the physical signs to which they gave rise. Nevertheless, I am sure you will find them of great interest. They consist of the heart and lungs removed *en masse*. On raising the sternum, the pericardium was found bulging, and, on opening it, a purulent fluid escaped. The pericardial layers, visceral and parietal, were covered, as you now see them, with a dense fibrinous exudate. About a pint (between four hundred and fifty and five hundred cubic centimetres) of seropurulent fluid was found in the pericardial sac.

The *left lung* was pneumonic from base to apex, and in the stage of red hepatization, there being no distinct attempt at resolution. It was firmly adherent to the chest wall along its anterior border, and also posteriorly; elsewhere covered with a thick exudate, which was also present on the corresponding portions of the parietal pleura. There was a considerable quantity of serous fluid in the pleural sac.

The *right lung* was united to the chest wall posteriorly by friable adhesions. Its pleural sac contained a considerable quantity of serous fluid. It was apparently the seat of compensatory emphysema, but otherwise healthy.

The other organs that were inspected presented no remarkable appearances. There were slight enlargement and congestion of kidneys, spleen, and liver, as well as congestion of the gastro-intestinal mucosa, and the consistence of the pancreas appeared to be unusually firm. The adrenals were normal.

The symptoms and physical signs, therefore, are exclusively correlated with left-sided pneumonia, pleurisy, and pericarditis, and this was the conclusion drawn from their study during life. The diagnosis was accurately made and the post-mortem findings were predicted. Such diagnosis may seem a simple affair, but the fact is that mistakes with reference to the presence or absence of pericardial effusion are constantly made. The late Dr. Fagge, one of the most accurate clinicians of his time, declared, in 1886, that "all the examples of very large chronic pericardial effusion which have occurred at Guy's Hospital have been set down to pleurisy." The case which I am discussing cannot be regarded as chronic, nor can the effusion be considered "very large." Nevertheless, it was considerable (more than a pint), and the complication with left-sided pneumonia increased the difficulties of the diagnostic problem.

I will now give you the principal facts of the clinical history. The patient was a young man, white, twenty-seven years old, born in the East Indies, and admitted to the hospital on March 10, 1902. Owing to his condition of extreme prostration, no attempt was made to obtain his family history. He was able to tell us, however, that he had been ill for the past ten days with pleurisy and pneumonia, attended by cough and bloody expectoration, pain in the left side of the chest, fever, and great weakness. His general appearance was that of a well-built, well-nourished young man. His decubitus was persistently left-sided. The face was of a dusky, cyanotic hue; there were marked dyspnoea (50 respirations per minute), a very rapid pulse (156), and a temperature of 99° F., the highest attained during his stay in the hospital. *Inspection of the chest* showed marked œdema of the superficial tissues of the left side, which extended longitudinally from the clavicle to the crest of the ilium and laterally from the left border of the sternum to the pos-

terior axillary line of the same side. The breathing was rapid, shallow, and labored. The respiratory movements of the left side were almost suppressed, while those on the right were exaggerated.

Physical Examination of the Lungs.—The left lung at its upper portion gave signs of consolidation,—viz., increased vocal fremitus, impaired resonance, and bronchial breathing, with a few fine crepitant râles. Over the base of the same side, anteriorly as high as the eighth rib, posteriorly as high as the seventh, there was diminished vocal fremitus, as well as flatness on percussion, while auscultation revealed distant bronchial breathing and distant bronchophony. In a dome-shaped area bounded above, in the midaxillary line, by the eighth rib, and before and behind by the anterior and posterior axillary lines, *there was marked, clear, and deep tympanitic resonance.* The respiratory movements of the right side were, as already stated, exaggerated, and the breathing was puerile; otherwise nothing abnormal was detected.

Examination of the Heart.—The apex beat of the heart was neither visible nor palpable. The precordial dulness merged for the most part into that of the consolidated lung, but extended about one inch to the right of the right sternal border. The heart sounds were distant and muffled, but free from murmur.

March 11.—As is the rule in all acute cases in this hospital, the temperature, pulse, and respiration were recorded every three hours. Eight such observations were, therefore, made between midnight of March 10 and the same hour of March 11. The highest recorded temperature of this day was 98.4° F., at 8 P.M.; the lowest, 97°, at 11 P.M. The pulse ranged between 164 at 5 P.M. and 110 at 11 P.M., and the respirations between 50 at 8 A.M. and 36 at 11 A.M. An exploratory puncture was made in the eighth intercostal space and a small syringeful of serous fluid withdrawn, but no fluid was obtained by the introduction of the trochar.

The tympany in the left axillary region above referred to was so pronounced that suspicions of pneumothorax were excited. No succussion splash could, however, be obtained, nor could the coin sound be elicited.

March 12.—The patient appeared to be more comfortable, the pulse and respiration being decidedly slower. The former ranged between 120 at 2 A.M. and 104 at 8 A.M., 5 P.M., and 8 P.M. The temperature ranged between 98.2° F. at 2 A.M. and 97° at 2 P.M.

and 11 P.M. With a small exploring needle introduced in the eighth intercostal space, a small quantity of fluid was withdrawn, as on the preceding day. A trocar was then inserted, but, as before, in vain. It was evident that the trocar came into contact with the lung and that there was little or no fluid in the pleural sac.¹

Notwithstanding the subjective improvement and the apparently more favorable ranges of pulse and temperature, the cyanosis continued unabated and the œdema increased. The finger could fairly be buried in the water-logged tissues of the thorax. The left lateral decubitus persisted, the patient being unable, even for an instant, to lie on his back. It appeared evident that such intense dyspnoea and cyanosis could not be accounted for by unilateral pulmonary consolidation, and it was concluded that the area of tympany in the left axillary region was the result of pressure and strictly analogous to Skoda's resonance at the apex of the lung in cases of pleurisy with large effusion. The diagnosis of pericardial effusion complicating left-sided pneumonia, previously hazarded, was now confidently pronounced.

March 13.—The ranges of pulse, respiration, and temperature were practically the same as on the 12th, but the patient was somewhat delirious, more restless, and evidently weaker. At 3.30 P.M., when I visited him, I introduced an exploratory needle into the pericardium through the fourth intercostal space about one inch to the left of the left sternal border, but obtained no fluid. In order to perform the operation, it was necessary to turn the patient, who was lying almost on his face, and slightly to raise his trunk. He expired almost simultaneously with the introduction of the needle. I naturally supposed that I had punctured his heart, but at the autopsy there were no signs of such an accident, which, besides, was precluded by the large amount of fluid in the pericardial sac. The failure to obtain fluid from the pericardial sac was attributed to the density of the fluid in that cavity.

A study of this case has convinced me that the diagnosis of pericardial effusion complicating extensive left-sided pneumonia is to be made as much by the symptoms as by the physical signs. It is impossible in such a case to outline the area of precordial dulness, and the projection of the right side of the heart beyond the right

¹The fluid found *post mortem* was undoubtedly a subsequent, præ-agonal accumulation.



Left-sided pneumonia with purulent pericardial effusion. A, cut surface of diseased lung; B, external surface of diseased lung; c, pericardial exudate.

border of the sternum might as reasonably be attributed to engorgement and dilatation of the right ventricle as to pericardial effusion. The muffling and weakness of the heart sounds may be no greater in pericarditis with effusion than in cases of pneumonia in corpulent individuals. On the other hand, the intense dyspnœa, the cyanosis, and the persistent left lateral decubitus should excite suspicions of something superadded to pneumonia,—in other words, of pericarditis. There was, however, in this case one physical sign which is at least alluded to by all systematic writers, but upon which none, in my opinion, lay sufficient stress. I refer to the marked tympany in the axillary region, which I have stated to be analogous to the tympany of Skoda's resonance. There is, I acknowledge, one objection to this comparison. In Skoda's tympany the lung at the apex is compressed to a certain extent by the upward pressure of the pleuritic effusion, but it is lung that has not been *consolidated* by previous inflammation. The tympany of Skoda is ascribed to "relaxation by compression." In the case I am describing to you the lung compressed by the adjacent distended pericardium was already consolidated. It may be argued that the tympany in this case was simply that of pneumonia independent of any outward pressure, for, as is well known, a marked tympanitic resonance is sometimes obtained over pneumonic foci. Inasmuch, however, as tympany in the left axillary region is generally recognized as a physical sign of pericardial effusion, it seems to me more reasonable to regard it as induced by pressure in this case, or, at least, as intensified thereby.

The question whether immediate incision and drainage of the pericardium would have saved this patient's life must have arisen in your minds, as, I need scarcely say, it has in mine. I have no hesitation in giving it a negative reply. The administration of ether would, I believe, have precipitated death, and the shock of operating without ether would probably have been equally fatal. In fact, the necessary change of position incident to either procedure would, in my opinion, have caused fatal syncope.

The nature of the inflammatory processes in this case was not precisely ascertained, although the fluid withdrawn from the pleural cavity on March 11 contained pneumococci.¹

¹ I am indebted to Dr. Adèle Russell Emerson, of Boston, for the excellent water-color sketch from which the plate illustrating this lecture was made.

DIFFERENTIAL DIAGNOSIS BETWEEN TUBERCULOSIS OF THE LUNGS AND DISEASES WHICH RESEMBLE IT.

BY LAWRENCE F. FLICK, M.D.,

Of Philadelphia.

TUBERCULOSIS of the lungs is simulated in its symptomatology by right-sided heart disease whether valvular or muscular, empyema, cirrhosis of the liver, cancer of the lungs, mediastinal, diaphragmatic, or intra-abdominal abscess emptying through the lungs, and syphilis of the lungs.

Right-sided heart disease, with the complications which usually accompany it, may resemble tuberculosis so closely that it is practically impossible to distinguish the two. This, of course, is true only in extreme cases, for there is always the possibility of making a diagnosis by the tuberculin test. Cases arise, however, in which the tuberculin test is inapplicable because of the advanced condition of the heart trouble. Right-sided heart disease without complications is easily enough recognized, and so is tuberculosis when not complicated with heart disease, but when right-sided heart disease is complicated with chronic bronchitis, as it often is, and when tuberculosis of the lungs is complicated with organic heart disease, the symptoms of the two conditions are so nearly alike that they cannot be distinguished. In a right-sided heart disease we always have engorgement of the lung tissue with varying degrees of œdema, and in long-standing cases we frequently have hypertrophy of the bronchial lymphatic glands with bronchorrhœa and thickening of the bronchial mucous membrane. Persons suffering from this condition are prone to catching cold and rarely are free from chronic cold. Streptococcus infection of the mucous membrane sets in, so that we may have occasionally some elevation of temperature; because of a dilated heart we have increased pulse-rate; we are apt to have profuse expectoration; we have persistent cough; owing to the general interference with the nutritive system due to the heart trouble, we have impaired nutrition; we sometimes have sweats;

frequently we have anæmia; we always have prolongation of the expiratory murmur, and sometimes we have impairment of resonance. This symptomatology gives a striking picture of tuberculosis because it contains practically all the symptoms which we find in chronic tuberculosis. There is some difference, however, between the complete symptom-complex of a right-sided heart disease and that of tuberculosis. In right-sided heart disease, with bronchitis and enlargement of the bronchial glands, we usually have prolongation of expiratory murmur over both lungs and over all parts of them in the same degree and with a striking evenness. Impairment of resonance is always evenly distributed and never amounts to complete dulness. There is apt to be perfect resonance above both clavicles and high up over the apices back. Impairment of resonance extends evenly along both sides of the vertebræ, forming a perfect tree, as the bronchi extend on both sides into the lung tissue, with greatest impairment at the roots of the lungs alike on both sides. When right-sided heart disease is uncomplicated with thickening of the mucous membrane of the bronchi and enlargement of the bronchial glands, and is only accompanied by œdema of the lungs, we are apt to have subcrepitant râles in one or both bases of the lungs with fair respiratory murmur in the apices, except, possibly, some prolongation of the expiratory murmur, with good resonance over both upper lobes and slightly impaired resonance over one or both bases. The subcrepitant râles in these cases is quite distinctive, conveying the impression that there is something sticky in the bronchioles which prevents the air-cells from opening freely.

In contrast with these physical signs of right-sided heart disease we always have, in tuberculosis of the lungs, physical signs which point to a localized condition, circumscribed and clearly definable wherever situated. In the early stages of the disease, even when complicated by cold and bronchitis, we can outline the infiltration by the impairment of resonance, absolutely distinct and discernible from any impairment of resonance that may be produced in other parts of the lungs by transient causes. When the disease is in one apex, without complication of a cold, there is prolongation of expiratory murmur or feeble respiration in that apex, with impairment of resonance over the upper portion of the scapula and between the scapula and vertebræ in the back and above the

clavicle in front, while normal respiratory sounds can be heard over the other apex. When the tuberculous process is complicated with a cold, there is prolongation of expiratory murmur on the healthy side as well by reason of bronchitis, but normal resonance. When the tuberculous process is advanced, extending through an entire lobe, and possibly through an entire lung, with involvement of the root of the other lung or the apex of the other lung, we are still able to outline the diseased area by the percussion note and by the sharp contrast between the dulness over the diseased parts and the high resonance over the healthy parts. There are cases, however, which are so complicated as to make a diagnosis by physical signs and clinical symptoms extremely difficult and unreliable. In these the microscope is the arbiter, for when we find tubercle bacilli in the sputum we have absolute proof of the existence of tuberculosis, and when, after repeated examinations in cases in which there is profuse expectoration, we fail to find tubercle bacilli, it is fair to presume that the disease is not tuberculous. Of course, it is necessary that the bacteriological findings harmonize with the physical condition. In other words, if we have a case in which there is localized consolidation, as indicated by dulness over certain parts when all the rest of the lung is clear, and we have the clinical symptoms of tuberculosis, we may unhesitatingly make the diagnosis of tuberculosis whether we find tubercle bacilli or not, even though there be considerable expectoration and the expectorated matter has not the appearance of broken-down tissue. If, on the other hand, we have the clinical symptoms of tuberculosis with profuse expectoration but clear resonance over every part of the lung, with a granulated reddened pharynx, showing a chronic inflammation of the mucous membrane, with a right-sided heart disease, and with absence of tubercle bacilli in the sputum after repeated examinations, we may conclude that the case is one of right-sided heart trouble, and not of tuberculosis, no difference how typical the sputum may look as the sputum of tuberculosis.

In these cases one must not be too ready to arrive at conclusions, however, nor depend upon a few examinations of the sputum, for tuberculosis of a very benign character may complicate right-sided heart disease, in which condition the symptoms pathognomonic of tuberculosis may be absent, and right-sided heart disease may be

accompanied by stasis in one or both apices, which gives rise to all the physical signs of tuberculous infiltration.

The recital of two cases illustrating these conditions will probably give a better picture than any amount of description: A. T., a young woman, thirty-five years of age, gave a history of ill-health covering several years; she had had a cough, which never entirely disappeared, but became worse at intervals—sometimes accompanied by expectoration, at other times free from expectoration; occasionally she had had hæmoptysis; she had loss of weight, occasional night sweats, prolongation of expiratory murmur in both apices, fine, moist râles in one apex, impairment of resonance—amounting to dulness on one side—in both apices, rapid pulse, but fair heart action, except that the second sound was accentuated, and with the general appearance of failing health. This young woman was diagnosed as suffering from tuberculosis by reason of the symptom-complex just related, and was admitted to a sanatorium to be treated for her condition. Her heart condition had been overlooked, probably because the symptoms were not marked at the time, and she was sent to the country at an elevation of about eight hundred feet above sea-level. The heart dilated and she became dropsical. Under treatment the dropsy disappeared, and for a while her general condition improved. She was then removed to an elevation of about fifteen hundred feet above sea-level. Dilatation of both sides of the heart set in, with hypostatic pneumonia and general dropsy, and after a few weeks the patient died. Dulness over the apices in this case continued to the end, hemorrhages occurred at intervals, and profuse expectoration took place, but at no time were tubercle bacilli found. To the end the case remained a doubtful one, although the probabilities are that it was a case of heart disease and not of tuberculosis. Unfortunately, an autopsy was not obtainable.

S. D., a married woman, thirty-five years of age, plump, well developed, and of healthy appearance, has had a cough varying in intensity for several years; has at times lost flesh and has again regained it; is at present of normal weight and in fair condition of health. Physical examination reveals slight murmur, with first sound of heart heard at apex, increase of area of heart dulness to the right, prolongation of expiratory murmur, distributed over both lungs evenly, slight impairment of resonance along the bronchial tree on both sides, no absolute dulness anywhere, and fair resonance

above both apices. Microscopical examination of the sputum reveals tubercle bacilli of a highly attenuated form, indicating low vitality of the organism and the benign character of the disease. This woman was diagnosed as a case of heart disease with a complication of bronchitis, and this diagnosis was justifiable until the tubercle bacilli were revealed by the microscope.

The two cases here recited are extreme cases, between which there are many leaning to the one side or to the other, but they well illustrate how difficult it may be to make a differential diagnosis between these two conditions.

Empyema is a pathological condition which closely resembles tuberculosis and is frequently mistaken for it, especially when the contents of the pleural sac have emptied into the bronchial tubes. In these cases there is a severe cough extending over a long period of time; there may be sweats; there is profuse expectoration, which has the appearance of tuberculous matter; there may be accelerated pulse-rate; there is sometimes slight elevation of temperature; there is dulness over the base of the lung and sometimes over the roots of the lung, on one side especially; there is prolongation of the expiratory murmur, most marked over the affected side; and there is normal resonance, as a rule, in all of the lung tissue except on the side where the empyema exists and along the sinus through which it empties. In almost every instance, however, there is resonance above the clavicle and over the upper portion of the scapula, showing that the apex of the lung is in normal condition, and this, when accompanied by absence of tubercle bacilli in the sputum after repeated examinations, is sufficient ground for a differential diagnosis.

Tuberculosis rarely begins in the bases of the lungs, and when it does the broken-down tissue shows the presence of tubercle bacilli as soon as the broken-down tissue is given off. Moreover, when tuberculosis does begin in the base of a lung, it is apt to extend rapidly to the apex of that side, so that it is unusual to have a tuberculous process advance to the breaking-down stage in the base of the lung without having an apical involvement. The pulse and temperature are usually normal in empyema, except when there is a complication of a cold or influenza, or when complicated with heart disease. The bodily weight has not been much reduced, considering the duration of the disease, and the general appearance of the

patient is that of fair health. In making a diagnosis, however, it should always be borne in mind that tuberculosis and empyema may coexist, and that it is not conclusive that tuberculosis does not exist because empyema is present. Repeated examinations of the sputum should be made in all such cases, and when the physical signs and bacteriological findings harmonize, it is safe to conclude that the case is one of empyema, and not of tuberculosis.

Hypertrophy of the liver is sometimes mistaken for tuberculosis, although it is difficult to understand how a careful man, with a proper knowledge of both conditions, can make such a mistake. When the liver is much enlarged and is pushed up under the diaphragm, and when there is a chronic bronchitis, kept up by alcoholism, accompanied by morning cough and expectoration, doubt may arise, but it is a doubt which can be easily cleared up by a microscopical examination of the sputum and a careful physical examination of the lungs. In such cases the lung on the right side is pushed up somewhat, and liver dulness encroaches considerably upon what ought to be lung resonance; but perfect resonance will be found in every other portion of the lung, which fully offsets the disturbing element of the prolongation of the expiratory murmur, which may be caused by the chronic bronchitis. Usually the subject in which this condition exists is well nourished, of normal and possibly supernormal weight, has a normal pulse-rate and temperature, and presents the appearance of fair health, except that he may be somewhat anæmic, languid, and dull. He is apt to complain a great deal, to be hypochondriacal, and to have the idea, in spite of all you can say to him, that he has tuberculosis. Usually he comes to you with his diagnosis made, and finds fault with you if you do not corroborate it.

Cancer of the lungs is a rare pathological condition, but when it exists resembles tuberculosis very closely. Quite naturally, all the subjective and objective symptoms are those which tuberculosis presents. As in tuberculosis, the diseased lung-tissue is circumscribed, easily outlined, and in sharp contrast with the healthy tissue. To a great extent the differential diagnosis must be made by the general condition, rather than by any marked individual symptoms. Usually the disease runs a very rapid course, involving a large amount of tissue in a very short period of time, and without culmination in softening. Although the patient will inform you that

he has been sick but a few weeks or possibly a few months, you will find the entire lung on one side, or perhaps two-thirds of the entire lung, involved, a severe cough, some fever, sweats, no expectoration except of a thin, mucal, purulent character, the respiratory murmur much better than you would expect to find, and absolute dulness over the diseased tissue. The lymphatic glands are apt to be involved, and may be easily outlined when they are. There are no tubercle bacilli in the sputum, and the general appearance of the patient does not correspond to that which is so typical of tuberculosis. In the early stages of the disease, a differential diagnosis is exceedingly difficult, if not impossible, unless it can be made by the tuberculin test.

Mediastinal, diaphragmatic, and intra-abdominal abscesses, when they empty through the lungs, may be temporarily mistaken for tuberculosis, but the diagnosis can usually be cleared up by a microscopic examination of the expectorated matter and the physical condition of the apices of the lungs. It is not always easy to locate the seat of these abscesses with precision, but when it can be made out that the upper portion of the lungs is entirely normal, and the outer rims of the bases on both sides are resonant, and there is absence of tubercle bacilli in the expectorated matter, no difference how much bronchial disturbance there may be for the time being, it is safe to conclude that the case is not one of tuberculosis. Such cases, moreover, run a rather acute course, and give a history of some acute disease which gives the key to the diagnosis.

Syphilis of the lungs is described in text-books and occasionally reported in journals. I have no personal experience with it. The differential diagnosis can, no doubt, be made by aid of the microscope and upon the history of the case.

From what has been said, it must be plain that the differential diagnosis between tuberculosis of the lungs and the diseases which resemble it is not always easy. In typical cases a mistake is not apt to occur, but in atypical cases the wrong diagnosis is frequently made. In order that one may guard against mistakes, the diagnosis should not be decided upon in any disease of the lungs until the entire subject has been studied from every possible point of view and every exact method of gaining information has been used. When this has been done, few mistakes will be made.

ABDOMINAL DIAGNOSIS.

BY E. STANMORE BISHOP, F.R.C.S. (Eng.),

Honorary Surgeon, Ancoats Hospital, Manchester; President of the Manchester Clinical Society; Fellow of the British Gynæcological Society, etc.

GENTLEMEN,—All patients with abdominal trouble complain of one of four definite things. Either they have (1) pain, (2) some swelling of the abdomen, (3) absence or abnormal modification of one of the normal excreta, or (4) some abnormal discharge. Any one of these primary features may be combined with one or more of the others or may stand alone, and the study of their relations and combinations is the only safe road to diagnosis of the conditions they indicate.

Symptoms and signs of disease may be divided into three classes,—first, those which are suggestive; second, those which are characteristic; and, third, those which are confirmatory. Of these, of course, the second group is of by far the greater importance; but such symptoms are often very hard to find, and indeed are frequently not present at all, so that we cannot always rely upon them: in which case we are compelled to fall back upon a combination of the first and third.

Our primary complaints, it will be evident, fall among those of the first class: they are suggestive only; a moment's consideration will show that any one of them is common to a large number of distinct diseases; but they are, after all, worthy of careful study. You will find that in the minds of those patients who present them they dominate everything else. For instance, if pain is present, it is most difficult to obtain information on any other point, so engrossed is the subject of it by this the only thing of apparent importance to him; but not only is this so, but each of these primary features presents certain peculiarities, the due appreciation of which will often carry the examiner some distance along the way towards identification of the pathological condition present. To attempt any satisfactory view of all these four in one lecture would be futile. To-day, therefore, I can only hope to show you what may be deduced from the first great symptom, pain.

Now, abdominal pain is of three kinds: it is either continuous, intermittent, or a combination of the two,—a continuous pain with intermittent exacerbations. Pain must be differentiated also from tenderness, about which I shall speak later. Pain is a subjective sensation, not requiring any interference by the examiner in order to elicit it. Tenderness requires pressure before it can be determined. True pain, then, may be continuous, intermittent, or a combination of both. In this region of the body intermittent pain indicates either neuralgia or some obstruction to the free flow of the contents of one of the four great tubular systems present in the abdomen, urinary, biliary, fecal, or, in women, genital series. It is always dependent upon the peristaltic contractions of one or other of these tubes. Continuous pain, on the other hand, means a pathological condition of some mesoblastic tissue. If this distinction be kept in mind, it becomes clear why in many cases there is a combination of both, or why pain commencing in an intermittent manner later becomes continuous with exacerbations. Take, for example, the passage of a calculus along the ureter. At first, if small enough, it passes along the canal, producing little or no pain; after a while, either from an alteration in its axis, which brings the long diameter of the stone across the lumen of the ureter, or because of additions to its size from crystallization upon it of some of the urinary salts, or, lastly, because it reaches a point which is less favorable to its passage, as one of the three points at which these calculi are usually arrested,—viz., just below the renal pelvis, just above the iliac crest, or at the vesical opening,—it becomes more or less fixed and forms an obstacle to the free passage of urine through the ureter. At once the peristaltic action of this tube becomes sensible to the patient. The tensely filled ureter is compressed by the action of its own muscular wall, and pain is produced. But muscular action is always temporary. After a while it subsides and the pain passes away, to recur again as soon as the muscular fibres recover and begin again to contract. A rough foreign body cannot long remain, however, in contact with normal tissues, subjected to repeated pressure of this kind, without producing certain changes which involve the mesoblastic walls themselves. Inflammatory swelling, hypertrophy, and possibly ulceration begin, and at once the pain becomes continuous, with, however, exacerbations during the peristaltic contractions still going on in

the altered muscular wall. A similar sequence of events may be noted in the intestine when the fecal circulation becomes impeded from any cause.

Should the pathological change, however, begin in the mesoblastic tissues themselves, pain if present is continuous from the first, and exacerbations of an intermittent type will exist or not, accordingly as those tissues involve one of the three tubular systems or are situated at a distance from them. This rule, the reason of which is, I think, self-evident, will often supply a clue to the line of investigation required, and, when the diseased condition itself is ascertained, will give some idea of the stage at which the examination has been made. The converse, however, is not true; certain cases where extensive disease exists, such as carcinoma of intestine, for example, have been observed where little or no pain was present up to a late stage.

But how are we to distinguish intermittent neuralgic pain from that produced by peristalsis? There are four distinguishing characteristics.

First. Neuralgic pain is felt in the abdominal skin. A slight touch is almost as effective as deep pressure for the purpose of intensifying it; indeed, firm pressure will sometimes, though not always, relieve it. Visceral pain is intensified in proportion to the pressure exerted, and a light touch does not evoke it. This statement, however, does not refer to the visceral *tenderness* which Head describes, and which will be considered later.

Second. Neuralgic pain is most marked in, and indeed confined to the course of, the abdominal nerves themselves. It can always be elicited by pressure over certain definite points,—viz., those at which the anterior, lateral, and posterior branches pierce the deep fascia, points which are well known and distinct. It is as marked at the back as it is in front, generally more so. Visceral pain is mainly felt in front, and usually comes on entirely independently of any action on the part of the examiner. (See, however, remarks on *locality* of pain.)

Third. The intermittent pain of neuralgia is sharp, darting. Visceral intermittent pain is paroxysmal, wave-like, gradually becoming more and more intense and then dying away.

Fourth. Neuralgic pain is increased by cold, by insufficient feeding, by anything which lowers the general vitality. Visceral

intermittent pain is increased by any stimulus to peristalsis of the tube affected.

The mode of onset of pain is of importance. In some forms of disease the commencement of pain is sudden, whether continuous or not. This occurs in acute intussusception, for instance: a child is apparently perfectly well at one moment, the next, screaming with pain, which is evidently sudden and intense. In perforation of intestine, rupture of gall-bladder, empyema, or ectopic pregnancy the onset is equally sudden; so also with twisting of a volvulus or the pedicle of an ovarian cyst, in strangulated internal or external hernia, acute peritonitis, or renal or biliary colic. In other cases its onset is gradual. The pain of cancer, of an ectopic pregnancy before rupture, of gastric ulcer, duodenal ulcer, or dysentery, of distended bladder, of adhesions, of catarrhal appendicitis, of retroflexed gravid uterus, of pyosalpinx, metritis, or salpingitis, and of hydronephrosis or pyonephrosis often begins so gradually that it is impossible for the patient to say precisely when it first commenced.

Acute pain of sudden onset means a sudden impression upon healthy or comparatively healthy nerves; therefore it finds its most typical exemplification in renal or biliary colic due to the impaction of a calculus in an otherwise healthy ureter or biliary duct. Gradual pain points rather to a slow, insidious change in the tissues which very slowly and step by step affects their nerve strands, and its type is to be found in neoplastic changes, such as those of cancer. Inflammatory changes act in much the same way, though not so slowly; thus, the pain of empyema of the gall-bladder is comparatively gradual in onset compared with that of biliary calculi, but more rapid than that of hepatic carcinoma. The pain of cystitis is not sudden in its commencement, as is that of vesical calculus, and the pain of a vesical calculus in a bladder which has undergone inflammatory changes previous to its formation cannot be so clearly dated as the commencement of the pain of a calculus which has suddenly found its way into a healthy bladder.

Much, doubtless, of the *intensity* of such pain depends upon the kind of calculus present; thus, the spicular, hard surface of a calculus composed of oxalate of calcium produces much more suffering than the smoother coat of one formed by ammonium urate or the softer phosphatic stone; but this is a question of intensity, not of mode of onset.

With regard to the *locality* of pain, several statements have been made, which are doubtless true, but the emphasis that has been laid upon them is, I think, misleading. It cannot be doubted that the results of surgical exploration tend towards greater security, greater certainty, in the recognition of the relation of pain in certain regions to disease of certain definite viscera; thus, pain on pressure over McBurney's point when taken in conjunction with other symptoms almost always justifies belief in the existence of inflammatory changes in the appendix, whilst Mayo Robson has lately shown that disease affecting the gall-bladder produces pain which may be elicited by pressure over another point one inch above and to the right of the umbilicus. Occasionally cases do occur where pain is complained of on one side of the abdomen when the cause has been located on the other; but these cases are rather surgical curiosities, the reason of which we do not yet know, but which probably will sooner or later be found to be subject to some definite rule. Meanwhile it seems a mistake to emphasize them, as is so often done, until the student comes to look upon them as exemplifying the normal condition of things, rather than as being, what they are, at present unexplained exceptions, and his ideas become confused in consequence.

But the pain educed by disease of abdominal viscera is often referred to other regions than those immediately over the organ affected. Here, however, we are on far firmer ground. Whether or not this reference takes place through certain definite segments of the spinal cord, as Head believes, the relationship is a matter of constant and consistent experience, and at once gives us a clue upon which we may rely. It is not based on isolated instances which appear to be contradictory to previous experiences, such, for instance, as the pain at the inner side of the knee complained of by women with metritis, or the pain at the shoulder in hepatic disorders.

The combination of *pain* with *tenderness* is of great value. It enables us to distinguish ordinary or lead colic, in which there is severe pain, but no tenderness, from peritonitis or appendicitis, in which both are present; gall-stone colic or urinary colic, with its recurring paroxysms of pain without tenderness, from empyema of the gall-bladder or perinephritic abscess, in which both pain and tenderness are well marked.

There is a form, however, of referred tenderness over certain areas, with maximum spots at a distance from the viscera involved, to which Head and others have called attention, which at first sight seems to invalidate to some extent the remarks in the last paragraph. But these areas are in relation with the viscera not by direct superposition, as is there assumed, but by and through certain spinal segments with which their sympathetic supply on the one hand and certain cutaneous nerves on the other are connected. These nerves are apparently not the ordinary spinal nerves of sensation, since each area crosses over the lines of several nerves, and lie much more transversely than does their line of distribution. Moreover, these areas do not overlap, as do the areas of common sensation. They appear to have much more in common with trophic nerves, and Head specially calls attention to the fact that the areas correspond with those mapped out by herpes zoster, when it occurs. They are tested, not by ordinary touch, but by the non-ability to distinguish between the touch of a blunt and that of a sharp instrument,—between the head and the point of a pin, for instance. They, therefore, do not perfectly resemble the usual idea of what is meant by tenderness, and might perhaps be better described as areas of hyperæsthesia. In this locality Head describes such areas from the fifth dorsal to the sacral areas. Following him, the various organs may be represented on the skin in the following way. (Figs. 1, 2, 3, and 4.)

Viscus.	Area.	Maximum Points.
Œsophagus, cardiac end...	Infrascapulo-mammary.	Anterior : fifth rib, one inch internal to nipple-line. Posterior : seventh spine, one and one-half inches from mid-line.
Stomach.....	Subscapulo-ensiform.	Anterior : over ensiform cartilage. Posterior : ninth spine, from this to scapular angle.
Liver.....	Middle epigastric.	Anterior : eighth space, two inches outside nipple-line. Posterior : one and one-half to two and one-half inches below angle of scapula, and two to three inches outside of mid-line.

FIG. 3.

AREAS. MAXIMUM POINTS.

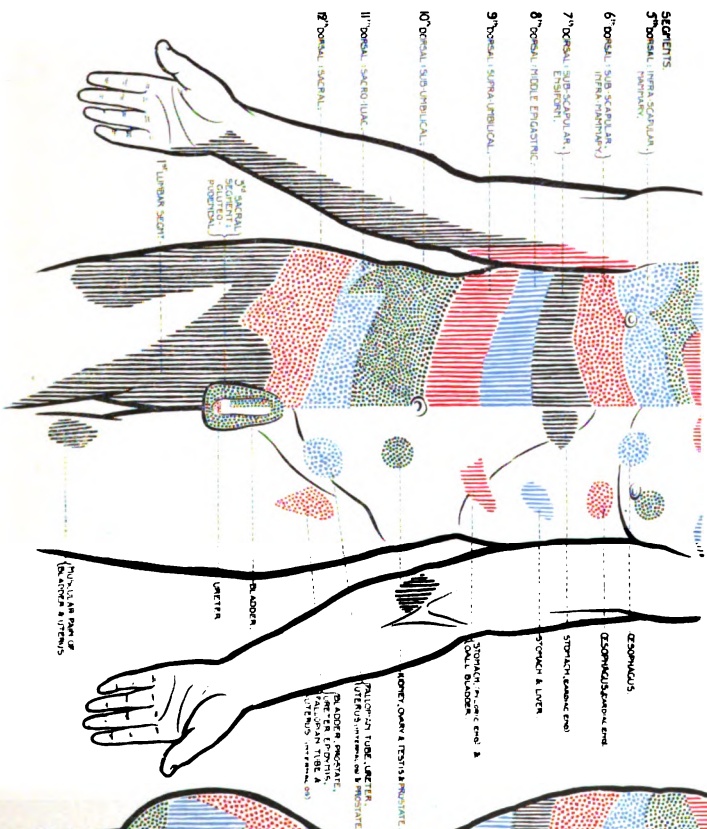
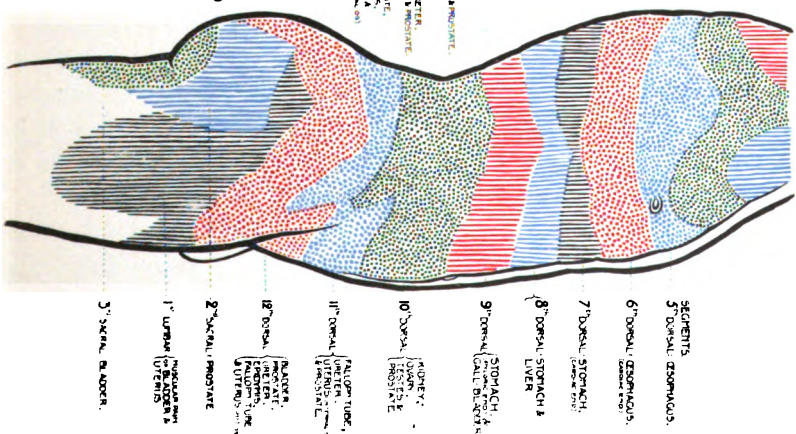


FIG. 4.

LATERAL AREAS.



Viscus.	Area.	Maximum Points.
Gall-bladder.....	Supra-umbilical.	Anterior : tip of tenth costal cartilage. Posterior : eleventh dorsal spine, one and one-half inches from mid-line.
Kidney.....	Subumbilical.	Anterior : one and one-half inches outside and one inch below umbilicus. Posterior : tip of twelfth rib.
Uterus.....	Gluteo-crural.	Close to great trochanter. Above and on inner side of knee.
Fallopian tubes.....	Sacro-iliac.	Anterior : above Poupart's ligament at level of internal ring. Posterior : fifth lumbar and first sacral spines.
Bladder and prostate.....	Sacral area.	Anterior : tip of glans penis. Posterior : over ischial tuberosity ; over lower sacrum.

The stomach, however, affected both its own and the area above if the lesion were at the cardiac end, or its own and that below if at the pyloric extremity.

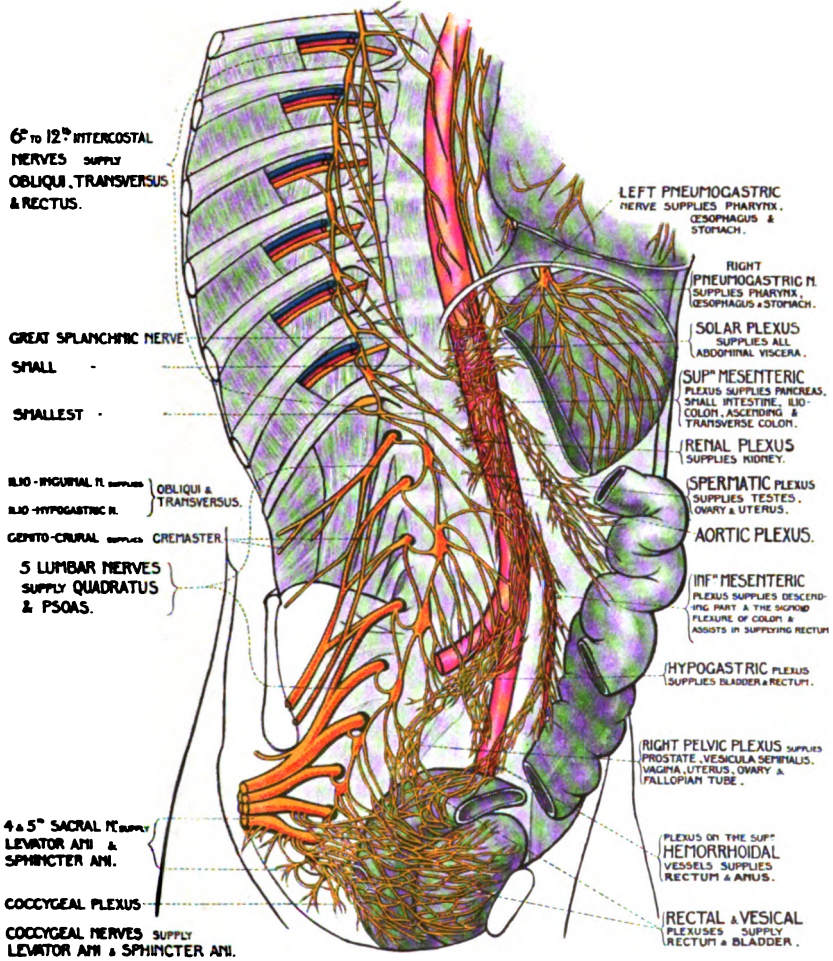
The tenderness of peritonitis is peculiar, since it has no posterior area, but extends laterally to a line stretching from the tip of the eleventh rib vertically to the crest of the ilium, beyond which it is not felt. Moreover, it emphasizes the difference between what is generally understood by the term tenderness and these areas of hyperæsthesia, since Head says that there is no true cutaneous tenderness in peritonitis, and that the tenderness undoubtedly present in this condition differs fundamentally from that described by him in connection with referred pain.

The combination of *pain* with *rigidity* is important. So long as the peritoneal surface of a viscus is not inflamed, so long rigidity will not be present; thus, a gastric ulcer may cause pain, but if it has not reached the peritoneal coat of the stomach there will be no rigidity. Catarrhal appendicitis will produce pain, but no rigidity unless there be as well some peri-appendicitis. But directly the peritoneum itself becomes involved, rigidity, local or general, according to the extent to which this membrane is implicated, will become evident. It appears to be another exemplification of the old axiom of Hilton, that irritation of the membrane lining a joint

is reflected at once to the muscles which move that joint. So in morbus coxa, rigidity of the hip-joint is the first and invariably present symptom. This rigidity of the abdominal muscles, however, is but temporary, and disappears after a while, the rapidity with which it does so appearing to have some relationship to the acuteness of the septic poisoning of the system which so rapidly supervenes in most forms of peritonitis. Thus, when distention begins, showing septic paralysis of the bowel, rigidity passes away; but I believe that it will always be found in the earliest stages if carefully looked for. The accompanying drawing, from Quain's "Anatomy," shows the intimate connection between the visceral nerves, derived from the sympathetic, and the motor nerves of the abdominal muscles. Quain says, "The sympathetic ganglia are severally connected with the spinal nerves in their neighborhood by short filaments; each connecting filament consisting of a white and gray portion, the former of which may be considered as proceeding from the spinal nerve to the ganglion, the latter from the ganglion to the spinal nerve; thus, the obliqui, transversales, and recti are supplied by the lower intercostal nerves, whilst the great, small, and smallest splanchnic nerves are formed by branches from the fifth to the eleventh dorsal spinal ganglia, and these splanchnic nerves supply the solar plexus, the semilunar ganglia, the suprarenal, renal, celiac, aortic, and other plexuses, from which the viscera are supplied. Other connections between the pneumogastric, the lumbar, and sacral nerves on the one hand, and the celiac, coronary, hepatic, superior mesenteric, hypogastric, and pelvic plexuses on the other are equally demonstrable." (Fig. 5.)

Lastly, *present acute pain* derives much of its significance from the history or want of history of previous subacute attacks,—their locality, duration, and character; thus, the acute pain of gastric perforation may sometimes be differentiated from that produced by duodenal perforation by the previous history of pain occurring within half an hour after the ingestion of food in the former as opposed to a history of such pain over two hours after food in the latter; whilst both may be distinguished from that of a ruptured ectopic pregnancy by the fact that the latter had a previous history of pain unconnected with meal-times, more persistent, and spreading down anterior crural, obturator, or sciatic nerves. This is useful, since the immediate picture is very much the same in all three.

FIG. 5.



CONNECTION BETWEEN SYMPATHETIC NERVES SUPPLYING VISCERA & SPINAL NERVES SUPPLYING MUSCLES OF ABDOMINAL WALLS.

• 92 •

SCARS AND MARKS OF CLINICAL INTEREST.

A CLINICAL LECTURE DELIVERED AT THE ARAPAHOE COUNTY HOSPITAL.

BY J. N. HALL, M.D.,

Professor of Medicine in the Denver and Gross College of Medicine.

GENTLEMEN,—It is of such great importance to you in your life work to cultivate the habit of careful observation that I shall devote this hour to showing you ten patients, regardless of their more serious illnesses, presenting some of the common scars and marks seen in daily practice. Scarcely a day will pass that you may not see similar instances, but many physicians overlook them entirely, or fail to realize their meaning if they are noticed. Do not understand that these little things are always of importance. It is because they are always interesting, and often of importance, that I call your attention to them. I wish emphatically to emphasize the fact that the hardest thing to learn in medicine is to see what lies plainly in view. It has been well said that “for every student who can see clearly what lies before him, ten can reason well upon the subject after seeing it.”

To illustrate: A short time ago I drew a circle two inches in diameter about the place of a visible apex beat in a cardiac patient, and asked the house physician to describe everything he could see within it. He mentioned almost everything, except the most striking point, viz., a well-developed supernumerary nipple. After I called his attention to its presence, he could not understand why he had not noticed it. In these matters it is often like seeing the man's face which may be found in some puzzle; it is hard to find, but wonderfully plain after it is detected. Many a physician has told me that he never saw a supernumerary nipple, yet if one hundred people would assemble at random, I doubt not but that the presence of this condition could be demonstrated in one or more of them. In fact, I often have two or three such cases in my wards at once. The presence of supernumerary nipples may be intrinsically of no importance, but I know of nothing that better illus-

trates our carelessness than the fact that we daily overlook so many points in our examinations. To become a good diagnostician, one must cultivate his powers of observation as assiduously as if he would be a detective.

The first patient shows the too-common marks of the hypodermic needle used in taking morphine subcutaneously. Those subjects who use the drug in this manner show bluish scars on the parts of the body and limbs accessible to both hands. This one utilizes the upper arms, forearms, and thighs chiefly. The breast, flanks, and legs are also frequently covered with these marks. They vary in size from two to four millimetres, and in color from a faint blue to dark bluish-black. The color is due largely to the pigment deposited by a needle which has lost its polish and has thus become coated with an oxide of iron. A new clean needle used by a physician leaves no appreciable mark. When these scars are abundant, you may almost certainly find the marks of abscesses scattered about among them. The morphinomaniac does not keep his syringe clean, and infection results. This patient shows not only the irregular scars where abscesses have ruptured, but marks of the surgeon's knife as well.

While our second patient possesses similar scars, they are mostly upon the chest and upper abdomen. Once, in seeking for œdema over the shins of an intimate friend of mine, I found just such scars upon the legs. Without observing them I should have been in ignorance of his drug habit.

The third case has a line of white, depressed, and shining cicatrices following the course of the eighth and ninth intercostal nerves upon the right side. They are somewhat irregularly aligned, but show here, as upon other parts of the body, a distinct tendency to follow some particular nerve. They often appear along the course of the lesser sciatic branches, occasionally on the brow or under the eyelids, and elsewhere. It is much better to ask such a patient when he had an attack of shingles than to ask him what made these scars. They are the result of ulceration of the skin in an attack of herpes zoster, while a similar herpes, under the heading "frontalis," "orbicularis," and so forth, accounts for the scars elsewhere. Like all scars, they in time become pale and a little less striking.

In our fourth patient are to be seen the ordinary scars of small-pox; as usual, chiefly situated upon the face. Upon the faces of

half of your patients you may find somewhere a single scar of similar nature. I see one now on the face of one of the nearest students, indicating that a chicken-pox vesicle has ulcerated and left its telltale mark.

Our next case has an exceedingly common form of scarring, which one might confuse with that of the last case. You will note, however, that it is chiefly upon the front of the chest below the clavicles. The white cicatrices, several hundred in number, are the results of suppuration from acne pustules healed many years ago. We find verification of this statement in the presence of several such acne lesions in an acute stage. The reddish-looking scars are much more recent than the pale, depressed, shining ones. These scars often appear upon the face, but more often over the upper chest and the scapular and interscapular regions of male patients.

Our sixth subject presents a less common form of marking upon the chest which you will see, perhaps, oftener in the female than in the male. Small, circular, white, slightly depressed, and rather evenly distributed cicatrices over the central upper region of the anterior thoracic wall indicate commonly that croton oil has been applied at some time for relief of thoracic trouble. It is generally easy to distinguish these marks from the others we speak of to-day by the characteristics just mentioned, and the absence of acne pustules. Smallpox and chicken-pox never have this peculiar distribution.

The seventh patient has a most interesting and not especially common form of scarring. Over the shoulders, neck, and arms, and less abundantly over other parts of the body, are innumerable fine, white, slightly sunken, and puckered scars, quite similar to those left by the croton oil in the last patient. These are the marks of a pustular syphilide of many years ago, for the true skin was damaged sufficiently to leave myriads of contracting cicatrices. In unconscious patients such marks may indicate to you something of the nature of the cerebral trouble.

In this man you notice a variety of cicatrices. They encircle the waist, and vary in size, shape, and color. Some are still red, while others are sunken and pale, indicating that they are older. These are the scars of ulcerating sores left by the irritation of the

too popular electrical belt. The peculiar distribution is practically pathognomonic.

The ninth subject is not my patient, but I show him to you through the kindness of Dr. Walbrach. I have asked him no questions, but will state to you that he has probably had, many years ago, an inflammatory affection of the right side of the chest, treated most likely by a German or Swedish physician. He has here and there the groups of six little parallel white scars so common upon patients of these nationalities above middle age. They indicate that wet cups were applied. The use of these appliances lingered longer among the Germans and Scandinavians than among other nationalities that we are called upon to treat. These marks are often found also over the lumbar region, indicating a diagnosis of nephritis or of lumbago; over the liver, stomach, and elsewhere, but less frequently in these situations than over the chest. They are scarcely ever seen in Americans. We will now ask the patient about them. He states that he left Germany when eighteen years of age, and he knows nothing about the cup-marks. They must have been made in infancy, and constitute much better evidence than we generally get from statements of patients with little intelligence or poor memory.

Our last patient presents so characteristic an appearance that any physician living in a coal-mining region ought to make a diagnosis as to occupation and disease at a glance. He has the barrel-shaped chest and cyanotic look of the patient with emphysema and chronic bronchitis, so common in those who inhale the irritating dust of mines. More characteristic are the small and rather faint bluish-black marks over the chest, arms, and back, indicating that here he has been struck by particles of coal as he has carried on his work, and that the carbon has thus become deposited in the skin,—virtually a tattooing of traumatic origin. Over the back and shoulders we often see much larger and more irregular black scars, the result of the falling of large lumps of coal from overhead. Often serious bony injury occurs at the same time, and especially fracture of the spine. These marks are very common upon the forehead and face, although not seen in this case. Quite similar scars from flying pieces of iron are seen in those who work about forges, but they are more apt to be confined to the arms and breast, and are generally smaller and less irregular. Scars from explosions

of the black blasting-powder, still used extensively in coal-mining, are of fairly uniform size and generally are quite numerous, and present the same aspect upon the body, having all occurred at one time.

In the patient presented as our sixth subject you may notice a puckered, depressed, rough scar an inch long over the most prominent part of the right scapula. It is the mark of a bed-sore occurring in an attack of pneumonia when he was sixteen years of age. Most scars of such origin are found over the sacrum, the trochanters, the scapulæ, and the heels, perhaps in this order of frequency. They are probably most common after typhoid fever, but rheumatic fever, pneumonia, cerebrospinal meningitis, and a few other conditions leave such scars in patients who recover. The serious bed-sores of persons with broken back, myelitis, and similar conditions do not generally heal, owing to the death of the patient.

Let us pursue the general subject a little further, for an immense variety of scars and marks will present themselves in your work. You will see the *lineæ albicantes*, not only as signs of a previous pregnancy, over abdomen and breasts, but in many other cases, and in the male sex as well. Over the hips and thighs of rapidly growing girls, and over the deltoid regions of boys who have fattened rapidly after the beginning of puberty, they are exceedingly common. Some have sought to explain similar *lineæ atrophicæ* following typhoid fever by a neuritis. I have never seen them until the patient began to fatten after his fever, and I see no reason for attributing to them any other origin than the rapid stretching from the gain in weight of a poorly nourished skin, which is less elastic than in health.

I have just seen a patient with scars in the groin which suggest the statement made in works upon medical jurisprudence for a century that such scars are to be taken as evidence of syphilis. This is certainly an error, as I have pointed out years ago. Suppurating buboes are practically unknown after syphilitic infection, while exceedingly common after other venereal diseases. They are to be taken as evidence against syphilis, excepting so far as one having them is shown to have been in the habit of exposing himself to venereal infection.

Upon the hands of old men who worked in flour-mills years ago, in the days of mill-stones, you occasionally see a most striking

form of tattooing. It was caused by the flying of pieces of steel and stone in the process of "picking" the mill-stone.

In this region you often see upon the street men who show, by the powder-marks in the right cheek, conjunctiva, and side of nose, that they have attempted to force a swollen shell into a breech-loading rifle. As the weapon is held on the right side of the body, the nose protects its own left side, which is commonly free from markings, although in the region of the left malar bone these reappear.

I shall merely mention the knife or tool marks upon the fingers of the hand with which one does *not* use the implement. These scars constitute far better evidence of right- or left-handedness, when found predominating upon the left or right hand, respectively, than most of the testimony commonly given in court upon such matters, as I have previously pointed out.

I shall, in closing, merely refer to the two near-by marks of an old seton, the scars of previous aspirations, of frost-bites, of old phlegmonous erysipelas, of cellulitis, of felons, of operations, and, if time permitted, of a hundred other conditions of interest. If you will keep your eyes open for all these things you will see as well, through your improved powers of observation, many a slight jaundice, abnormal pulsation, dilated pupil, or other sign of great importance in some obscure case.

THE NATURE OF CANCER AND OTHER NEW GROWTHS.

BY WILLIAM CECIL BOSANQUET, M.A., M.D. (Oxon.), M.R.C.P. (Lond.),
Physician to the Out-Patient Department at the Victoria Hospital for Children,
Chelsea; Pathologist to Charing Cross Hospital.

THE belief in a parasite of some kind as the cause of cancer appears so widely held at the present time, and the search for such an organism is being carried on with such intensity in laboratories all over the world, that it may be of use to consider briefly, upon general grounds, the inherent probabilities of such an organism being the true causative agent in the production of new growths, and the initial prospect of success in the pursuit. For this purpose it is necessary to consider the known facts with regard to parasitic or infective diseases, and to ascertain how far they are parallel with what is known of the phenomena of neoplasms. Since a new growth presents a distinct local lesion or lesions, it is with infective processes presenting local manifestations, rather than with general infectives, that it must be compared. The closest analogy, then, would appear to exist with morbid growths known as the infective granulomata, of which the best known is tuberculosis.

At first sight, there may appear to be a very close resemblance between the two conditions. There is in the case of a malignant growth a mass of new tissue which increases at the periphery and breaks down in the middle, gradually invading neighboring parts and causing their destruction; this is similar to a local tuberculous deposit. Again, the dissemination of cancerous nodules throughout the viscera, starting from the original lesion, is suggestive of the outbreak of general miliary tuberculosis which may result from an original focus. But, more closely considered, the two processes are very different—so different, indeed, as to point to the probability that very diverse causes are at work. The dissemination of infective material from the tuberculous focus results in the production, wherever the bacilli settle, of nodules due to multiplication of the cells of the connective tissue already existing in the part; in other

words, an inflammatory reaction is set up, the tendency of which, as of all inflammations, is to arrest the invading organisms and repair the damage done by them. In the case of cancer, on the other hand, the secondary nodules are formed by the growth of the actual cells carried away from the original tumor, which are implanted as emboli in the distant tissues; these cells act as parasites in their new surroundings, setting up inflammation in the neighboring parts, but the inflammatory material forms an inconsiderable fraction of the resulting nodule. If a parasite be the cause of cancer, it is necessary to suppose that it is carried on the embolic cells as a passenger and continues to stimulate the growth in their new quarters. This is not impossible; but the hypothesis cannot be supported on the ground of a resemblance to the recognized infective process, in which no transference of animal cells takes place, but pre-existing elements are caused to multiply by the irritation of a multiplying bacterium or its toxin.

In the case of tuberculosis, before the specific bacillus was discovered it was known that the injection of tuberculous material was capable of setting up the disease. Such inoculation does not appear to take place in cancer. Positive results have, indeed, been recorded in a very few instances, but such an occurrence is without doubt very rare. Even if such rare instances of inoculation be admitted, it is not thereby proved that the disease is due to a parasite; since, if an animal can inoculate itself by embolism with the cells of an original growth so as to suffer from secondary deposits, it is not impossible that inoculation with living cells may occur in another animal of the same species. The great variety of such transference would suggest the latter explanation, rather than an infective process caused by a parasite.

Again, it seems almost impossible on the hypothesis of a parasitic cause to account for the various forms of carcinoma—spheroidal-celled, columnar-celled, etc.—which are found under different circumstances. To suppose a different parasite in each variety is too great a stretch of the scientific imagination; to suppose that the same parasite causes different forms of growth according to its place of inoculation, these reproducing their special characteristics exactly in their secondary deposits, is scarcely more inviting.

Finally, it is necessary to bear in mind that new growths form a class by themselves, with sufficiently distinct general characters,

and that, therefore, it is *a priori* to be supposed that they are all due to similar causes. It is, no doubt, at first sight tempting to separate the malignant from the innocent or benign varieties and to attribute the former to a special cause; but there is not adequate scientific support for such a division. No hard and fast boundary line can be drawn between innocent and malign, since typically innocent growths (*e.g.*, chondromata, myomata) may under certain circumstances exhibit malignant characters, while neoplasms which have the structure of epitheliomata may at times (*e.g.*, in the palate) remain localized and benign. Now, it is only in carcinomata that any serious attempt has been made to prove the existence of parasites; even the most ardent upholders of the parasitic theory have not attempted to apply it to all new growths. Hence it must be looked upon with suspicion as tending to make of cancer a thing apart, taking it out of the pathological group to which it naturally belongs and assigning to it a special mode of causation.

It may be admitted that if it were once definitely proved that a special form of parasite occurred in all carcinomata and was found in no other condition, a strong *prima facie* case would have been made out for considering this organism to be the true cause of cancer. At present this is very far from being the case. The great majority of the parasites brought forward by different discoverers are merely appearances seen in sections of growth; they have not been isolated or cultivated, and are, without doubt, products of the degeneration of epithelial cells (hyaline, etc.). A certain number of observers have, however, cultivated fungi (blastomycetes) from cancers and grown them outside the body; but it is far from proved that these organisms are more than accidental contaminations. To prove their causal relation they must be reinoculated into susceptible animals and produce true cancer. No undoubted case of true epithelial tumor produced in this manner appears to be on record, and there is not at present satisfactory evidence of the invariable presence of an organism in all cancers or of the ability of any organism to produce such growths when inoculated.

It may be asked, then, to what mode of causation the phenomena of new growths seem to point. The nature of the metastases in malignant cases may perhaps suggest an answer. Since such secondary deposits consist of embolic cells carried away from an original focus, it is natural to suppose the latter also to have started

as a cell or group of cells, somehow displaced and cut off from its original connections. Such a severance might be effected in many ways. Direct traumatism is one way, and a certain number of patients attribute the origin of their disease to injury. A second mode of origin is the persistence of "rests," according to Cohnheim's well-known theory, certain cells having gone astray in the process of development. A third way is seen in the process of chronic inflammation,—chronic irritation and attempts at repair,—in which, as at the margin of a chronic ulcer, there are often found groups of epithelial cells cut off from the rest of the epidermis and lying as islands among the inflammatory elements. The origin of epithelioma for such a fragment is easily conceivable. In this connection it is interesting to remember the comparative frequency with which primary carcinoma of the liver—a rare condition—is found to have its seat in an organ already cirrhotic, the new fibrous tissue conceivably having cut off some group of cells from its fellows and started it on the road to independent growth. The coexistence of cancer of the breast with chronic interstitial mastitis may point in the same direction. On the other hand, it is possible that in such cases it is rather an irregular attempt at repair which results in the appearance of a tumor. A universal control of all the tissues by the nervous system is not, indeed, generally recognized; but trophic control of the muscular and glandular cells is conceded, and it is possible that connective tissues and epithelium are also under the same governance. Indeed, with regard to the skin it is scarcely possible to deny direct nervous influence, having regard to the proved facts relating to herpes zoster and the many other lesions connected with the distribution of nerves. If such a trophic influence does in reality exist, the loss of control over certain groups of cells may result in their growing in a luxuriant and riotous manner, without regard to the needs of the organism as a whole; in other words, in their forming tumors. It must, however, be admitted that such an hypothesis is difficult of verification. That neoplasms originate in groups of cells which are somehow enabled to take on independent growth remains a certain fact, whether this growth be stimulated by a parasite or be an innate quality of the cells themselves, or result from diminished central control.

SOME CLINICAL ASPECTS OF ANEURISMS OF THE AORTA.

AN ADDRESS DELIVERED AT THE ANNUAL MEETING OF THE RUTLAND COUNTY
(VERMONT) MEDICAL SOCIETY AND AT A MEETING OF THE GER-
MANTOWN (PHILADELPHIA) MEDICAL SOCIETY.

BY ALOYSIUS O. J. KELLY, A.M., M.D.,

Instructor in Clinical Medicine and Assistant Physician to the Hospital, Univer-
sity of Pennsylvania; Professor of the Theory and Practice of Medicine,
University of Vermont; Physician to St. Mary's and St.
Agnes's Hospitals, and Pathologist to the German
Hospital, etc., Philadelphia.

THE subject that shall engage our attention to-day is that of true aneurism as contrasted with false aneurism: true aneurism—one in which the aneurismal sac consists in whole or in part of the wall of the blood-vessel, as opposed to false aneurism—one in which the aneurismal sac consists of connective tissue, the wall of the blood-vessel having ruptured and permitted of the extravasating of blood into the surrounding connective tissue. With regard to the designations true and false aneurism, however, it is well to bear in mind that the conventional distinctions between them are more or less arbitrary, and that the sacs of many so-called true aneurisms, in their late stages at least, are composed almost exclusively of connective tissue. With the progress of the disease, the wall of the blood-vessel originally constituting the sac of the aneurism becomes atrophic, degenerated, and replaced by newly-formed connective tissue. To borrow the words of Hilton Fagge, then, "An aneurism may be defined as a circumscribed tumor containing fluid or solid blood, communicating directly with the canal of an artery, and limited by the tunic which is called the sac." Without discussing in detail the etiology of aneurism of the aorta, I have elected to say a word or two about several features of clinical interest that are of importance in themselves and that have a material bearing upon the causation of the affection.

Among these is the age at which such aneurisms develop. Such

is the prevalent opinion with regard to the dependence of aneurisms upon arteriosclerosis that one is likely at first sight to associate the incidence of aneurisms with the beginning of the fifth decade of life. Reflection, however, brings to mind the many cases of arteriosclerosis that arise before the fiftieth year, and examination of statistics proves that the largest number of aneurisms develop between the thirty-fifth and forty-fifth year of life. Thus Bizot found that of one hundred and eight aneurismal subjects almost as many were less than forty years of age as were between the ages of forty and seventy years. Crisp found that of five hundred and five cases, one hundred and ninety-eight occurred between the ages of thirty and forty years and one hundred and twenty-nine between the ages of forty and fifty years. Browne found that of one hundred and sixty-three cases, sixty-seven occurred between the ages of thirty-five and forty-five years and forty-seven between the ages of forty-five and fifty-five years. Hare recently, in a collective investigation, found that of six hundred and seventy-four cases, one hundred and fifty-two occurred between the ages of twenty-five and thirty-five years, two hundred and eighty-two between the ages of thirty-five and forty-five years, and one hundred and thirty-nine between the ages of forty-five and fifty-five years. Coats, directing attention to the fact that aneurisms are most frequent between the thirtieth and fortieth years, whereas atheroma is much more common after than before the fortieth year, aptly remarks that "aneurisms coincide with that time of life when the period of greatest bodily vigor overlaps the beginnings of the period of occurrence of atheroma." Additional interest is lent to this statement by the report from time to time of cases of sudden death due to ruptured aneurism in very young subjects,—even fifteen years of age.

Another interesting clinical feature of aneurisms of the aorta is the preponderance of males affected as compared with females. This, of course, is a fact that is generally recognized. The marked differences in the sex incidence, however, is well illustrated by certain statistics. Thus of sixty-three cases, Hodgson found fifty-six among males and but seven among females. Bizot found one hundred and seventy-one of a total of one hundred and eighty-nine cases among males and eighteen among females. Of five hundred and forty-four cases of sacculated aneurisms of the ascending portion of the arch of the aorta recently collected by Hare, four hundred and sixty-six

affected males and but seventy-eight females. These statistics collectively show that eighty-six per cent. of nine hundred and six aneurisms of the aorta occurred in males. This marked difference in the sex incidence is doubtless due in large part to the different environment in which man and woman live, but probably in part also to the greater abuse of different toxic agents, such as alcohol, etc., that obtains among men rather than among women. In this connection, however, it is well to bear in mind that, as pointed out by Coats and others, cerebral aneurisms occur with almost equal frequency in both sexes.

Then the question of the etiological relationship of syphilis to aneurism has been much debated. A large number of authors have for a long time held to the idea of a close causal connection between this form of infection and aneurism, and these have included not only competent clinicians, but also well known syphilographers. On the contrary, quite a number of observers have expressed considerable scepticism regarding this etiologic relationship. Of some recent writers that contend for the syphilitic nature of many cases of aortic aneurism I may mention Puppe, Hampeln, Kalindero and Babes, Malmsten, Heller, Straub, Gerhardt, Rasch, Drummond, and others. The statistics of these writers vary, however. Thus Rasch states that syphilis was certainly present in fifty-six per cent., and probably in eighty-two per cent., of the aneurisms studied by him; Malmsten places the percentage at eighty; while Drummond has "found it a good practical rule not to diagnose aneurism in a doubtful case in the absence of syphilis." Of those that oppose the idea of a marked etiological relationship between syphilis and aneurism I may mention Leyden, Ponfick, Ziegler, Orth, Gairdner, and others. The statistics recently collected by Hare also tend to show that syphilis heretofore may have been overrated as an etiological factor. Thus of five hundred and forty-four cases of aneurism of the ascending portion of the arch of the aorta no cause was assigned in two hundred and ninety-five; in but twenty-eight there was a history of previous syphilis; whereas forty-seven cases were assigned to traumatism, forty-five to alcoholism (three of these to traumatism in addition), seventeen to rheumatism, and four to syphilis and alcoholism combined. It would thus appear that we have overestimated the etiological importance of syphilis. We should bear in mind, however, that the evidence adduced is negative rather than

positive, that syphilis may really have been operative in a number of cases attributed to other factors.

To speak now briefly of the pathogenesis of aneurism, for the moment I cannot do better than to consider its purely physical characteristics. Quoting Coats, an aneurism may be said to be the result of "a local disproportion in the normal balance between the amount of lateral pressure inside the artery and the elastic resistance of the vascular walls, arising either from abnormal increase of the former or diminution of the latter; most frequently, indeed, from both these causes in combination." We have then to ascertain the causes of the diminution of the elastic resistance of the vascular walls on the one hand, and the causes of the increase of the lateral pressure inside the artery on the other hand.

The causes of the diminution of the elastic resistance of the vascular wall are intimately bound up with the causes of arteriosclerosis; in some cases the etiology of the one is really the etiology of the other. The intimate relationship existing between the two is sufficiently manifest from the presence of arteriosclerosis or atheroma in every case of aneurism that comes to necropsy. That, however, their relationship is not the simple one of cause and effect is apparent from the fact that, as already pointed out, aneurisms are prone to develop rather early in life, especially at the end of the third and the beginning of the fourth decade, whereas endarteritis or atheroma, as a rule, does not present itself until a later period, generally at about the beginning of the fifth decade; that is, aneurisms are common at a time of life when arteriosclerosis is uncommon; they develop, as has already been remarked, at "that time of life when the period of greatest bodily vigor overlaps the beginnings of the period of occurrence of atheroma;" whereas in advanced life, when arteriosclerosis is common, widespread, and of extreme degree, aneurisms are unusual. Furthermore, an aneurism, in its early stages at least, represents a local disease of an otherwise fairly or quite healthy vessel, and not an increase of the lesions of a diffusely diseased vessel. The interrelationship, then, existing between arteriosclerosis and aneurism may perhaps be fairly expressed by saying that both are due to like causes; that in one individual these causes result in the production of arteriosclerosis, whereas in another they result in aneurism,—the development of the aneurism being determined by a local predisposition of the vessel wall. Thus, while



FIG. 1.—Large aneurism of the transverse portion of the arch of the aorta. (From the Museum of the German Hospital of Philadelphia.)

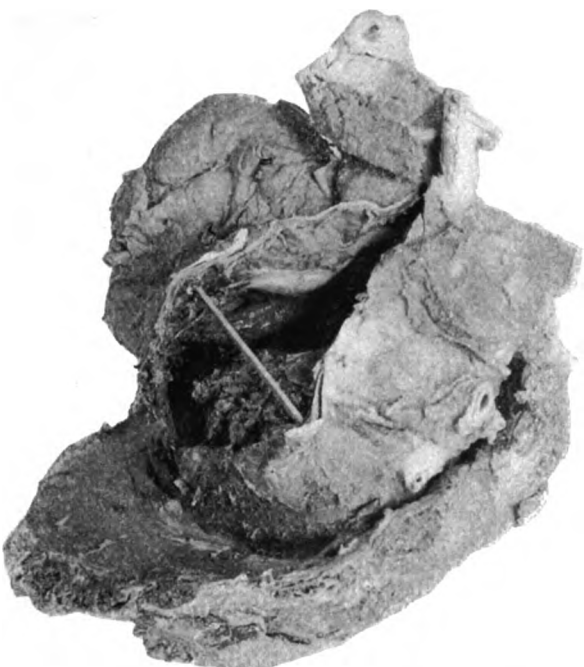


FIG. 2.—Large aneurism of the arch of the aorta with erosion of the sternum. (From the Museum of the German Hospital of Philadelphia.)



FIG. 3.—Double aneurism of the arch of the aorta, with sudden death from rupture into the esophagus. (From the Museum of the German Hospital of Philadelphia.)

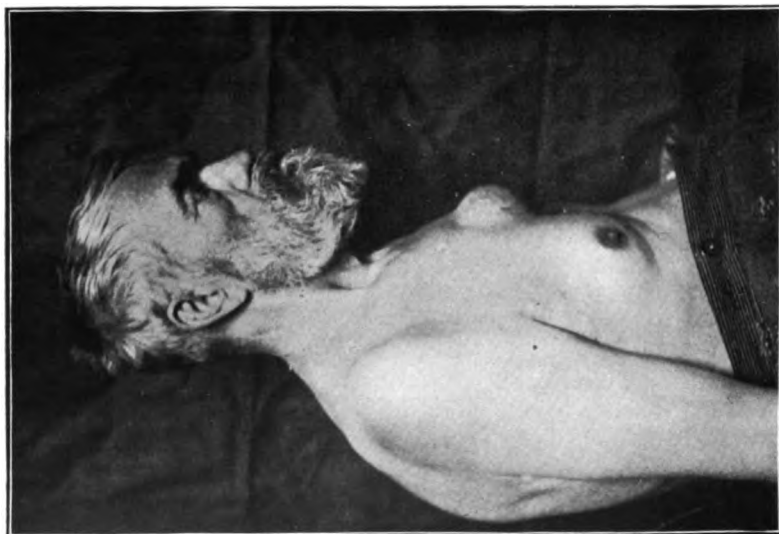


FIG. 4.—Aneurysm of the arch of the aorta protruding through the sternum. (See Figs. 5, 6, and 7.)



FIG. 5.—Aneurysm of the arch of the aorta protruding through the sternum. The patient shown in Fig. 4, one year later. Death occurred suddenly from external rupture through the skin. (See also Figs. 4, 6, and 7.)

there can be no question that the elastic resistance of the vessel wall is diminished in arteriosclerosis, when we have attributed all that we can to the etiologic influence of arteriosclerosis, we are still forced to assume as a factor in the pathogenesis of aneurism a local predisposition of the vessel wall. This local predisposition may be either congenital or acquired; it may be an inherent, localized weakness of the vessel wall, or an acquired deficiency of vitality the result of endarteritic alterations that may have developed, possibly during fetal life, or more likely during infancy or childhood,—the result of some of the acute infective diseases. In this connection it may not be inopportune to point out that any one with necropsy opportunities may almost any day assure himself of the not infrequent occurrence of small circumscribed areas of endarteritic alterations, of foci of arteriosclerosis, in adolescents.

However acquired, this local predisposition of the vessel wall finds its expression in a localized loss of tone or of elasticity. The first anatomic and physiological changes in the vessel wall in aneurism, as well as in arteriosclerosis, have long been the subject of much discussion and investigation. On the one hand, a small number of writers still maintain that the changes in the media are the result of primary disease of the intima. This view, however, is now generally abandoned,—the result in large measure of recognition of the painstaking researches of Thoma and of the now general use of specific elastica stains. The evidence at present at hand warrants the statement that the first changes in the vessel wall consist of alterations in the elastic lamina or the muscular coat or both,—structures that furnish both support and resiliency to the vessel. These changes, especially as now conceded, changes in the elastic membrane, result in dilatation of the vessel. Consequently there ensues a hyperplasia of the intima that serves to some extent as a compensatory measure tending to restore the lumen of the vessel to its normal caliber. Without entering upon a discussion of the many questions that obtrude themselves for consideration, it suffices to say that the importance of these mesarteritic, as well as of periarteritic processes in the pathogenesis of aneurism of the aorta is beyond question. As pointed out by Rosenbach, they hold the same relation to the development of aneurism that myocarditis and pericarditis hold to the development of dilatation of the heart. As further pointed out by Rosenbach, the prime importance of the

alterations in the median coat must be evident when we consider that the resistance of the arterial wall and the variations in its caliber are almost exclusively dependent upon the thickness and strength of its muscular coat and elastic lamina. That the intima itself cannot be of preponderating importance is plain from the fact that, under both normal and pathological conditions, the resistance that it can offer to the blood stream, as contrasted with other causes increasing blood pressure, is so slight that it is not possible that diminution of its functional activity, in the presence of the normal activity of the median coat, can lead to circumscribed dilatation of the vessel.

As a common cause of increase of arterial pressure, and as an important factor in the etiology of aneurism of the aorta, I wish now to allude briefly to prolonged and to sudden strain,—a subject to which Clifford Allbutt some years ago directed marked attention. In estimating the etiological significance of strain one must take into consideration all the factors in the individual case; and one may no more cursorily and without investigation attribute to strain certain manifest diseased conditions in a patient than he may elect to overlook the possible etiological importance of a sudden and severe strain to the influence of which the patient himself may attribute the onset of his ailment. It is quite conceivable, indeed it is quite true, that the healthy individual—the individual with his muscles, heart, blood-vessels, and lungs developed proportionately to the demands that he makes upon them—may endure a considerable amount of prolonged or sudden strain without obvious ill consequences. This we observe almost every day in athletes and like persons. But we also observe very frequently the disastrous results to the heart and the general economy of sudden severe strain and of prolonged strain ill-advisedly persisted in, not only in a certain proportion of those engaged in athletic contests, but also in those whose occupation necessitates considerable strain,—thus men rather than women, blacksmiths, iron-workers, draymen, etc., as well as in those poorly endowed by nature, who soon use up their reserve strength. Generally the first manifestations of prolonged strain consist of irritability of the heart,—palpitation, dyspnoea, increased pulse-rate, etc., but sooner or later myocardial alterations and dilatation with or without hypertrophy result. Allbutt maintains that the usual consequence of such dilatation, whether or not it be partially com-



FIG. 6.—Cast made after death from external rupture of the aneurism illustrated in Figs. 4, 5, and 7. The projection through which the fatal hemorrhage occurred is well shown. (From the Museum of the German Hospital of Philadelphia.)



FIG. 7.—Large aneurism of the arch of the aorta protruding through and causing almost complete absorption of the sternum and adjacent costal cartilages. (See also Figs. 4, 5, and 6.) (From the Museum of the German Hospital of Philadelphia.)

pensated, is chronic inflammation of the aorta and aortic valves, and that it is largely a matter of chance whether the valves first become incompetent or whether the aorta first becomes diseased,—the result of the continuously recurring impact of an excessive amount of blood forcibly projected against it with each systole of the heart. The disease that thus results from strain may consist either of loss of elasticity and dilatation of the aorta or of “points of endoarteritis, with diffuse granular exudation among the fibres of the middle coat,” followed by “pouching of the aorta, with consequent or concomitant incompetence of the valves.” “If, however, one sudden effort be the cause of the mischief, we never find pouching of the aorta as a consequence; but we find a crack, which may be in the floor of the aorta causing regurgitation, or in the side of it giving rise to saccular aneurism.” Not at present to pursue this important question any further, it suffices to point out the importance of sudden and of prolonged strain in the etiology of aneurism and to draw attention to the fact that once initiated the progress of the disease is much accelerated by strain.

With regard to the situations of aneurisms of the aorta, it is well known that the relative frequency of occurrence follows the course of the vessel,—that is, the greatest number involve the ascending portion of the arch, the next greatest number the transverse portion of the arch, then the descending portion of the arch, and finally the remaining portion of the aorta. In Crisp's collective investigation of two hundred and thirty-four cases of aneurism, the thoracic aorta was involved in one hundred and seventy-five (74.5 per cent.). Of one hundred and sixty-seven of these cases, ninety-eight (58.8 per cent.) involved the ascending portion of the arch, forty-eight (28.7 per cent.) the transverse portion of the arch, and twenty-one (12.6 per cent.) the descending portion of the arch. In Gibson's investigation of eight hundred and eighty cases, he found that the sinus of Valsalva was involved in seventy-eight cases, the ascending portion of the arch in one hundred and forty-one (exclusive of fifty-two cases of dissecting aneurism of this portion of the vessel), the transverse portion of the arch in one hundred and twenty, the ascending and transverse portions of the arch conjointly in one hundred and twelve, the descending portion of the arch in seventy-two, the transverse and descending portions of the arch conjointly in twenty, the entire arch in twenty-eight, the descending portion

of the aorta below the arch in seventy-one, the abdominal aorta at the celiac axis in one hundred and twenty-one, the abdominal aorta below the celiac axis in twenty-six, and the branches of the abdominal aorta in twenty. Of Hare's cases, five hundred and seventy involved the ascending portion of the arch, one hundred and four the transverse portion of the arch, one hundred and ten the descending portion of the arch, and in one hundred and sixty-nine cases the situation of the aneurism was not accurately defined.

It being inappropriate in this connection to discuss the morbid anatomy of aneurisms of the aorta, it will suffice to say that they may be single or double (rarely multiple), and that they may vary much in size and conformation. They may be very small, scarcely more than a slight bulging or dilatation of the vessel, or they may attain the size of a child's head. The accompanying illustrations will serve to show some of the more common appearances of such aneurisms.¹ (Figs. 1-8.)

The varying clinical manifestations of aneurisms of the aorta have been pointed out from time immemorial, and it is now well known that such an aneurism may be entirely latent, giving rise to no symptoms for a long time,—in some cases not until the fatal termination is induced by sudden rupture and hemorrhage. In perhaps the majority of cases, however, sooner or later the aneurism manifests itself by both subjective and objective symptoms. In the one case the subjective symptoms in the other case the objective symptoms are the more obtrusive or exist alone, the aneurism of symptoms, and the aneurism of physical signs respectively. Broadbent, to whom this convenient classification is attributed, says further that the aneurism of symptoms arises from the transverse arch, and the aneurism of physical signs from the ascending arch of the aorta.

The detection clinically of an aneurism of the aorta may be very easy, or it may be attended with unusual difficulties, being in some cases almost, if not quite, impossible. When we find in some part of the thorax (generally to either side of the sternum above the third rib, but sometimes even posteriorly) a pulsating tumor which is not the

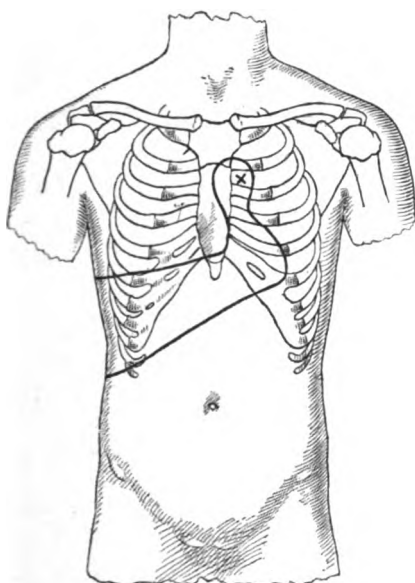
¹ For the photographs I am very much indebted to M. I. Wilbert, Ph.D., chief apothecary and director of the Laboratory of Photography and Radiography of the German Hospital of Philadelphia.



FIG. 8.—Large dissecting aneurism of the abdominal aorta. (From the Museum of the German Hospital of Philadelphia.)

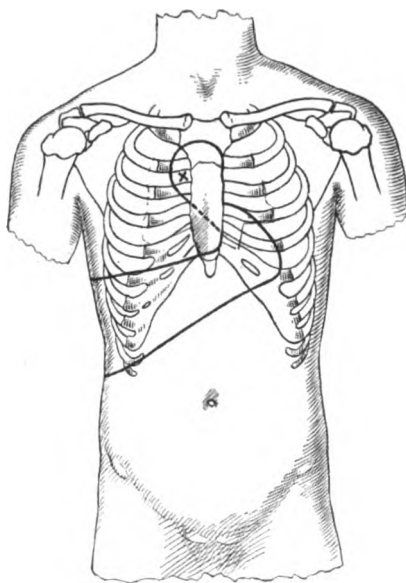
heart, although it beats isochronously with it and at least as forcibly, which at each pulsation expands in all directions, and over which there is dulness on percussion and a systolic or a systolic and a diastolic murmur, the diagnosis of aneurism, even in the absence of other signs or symptoms, is of course warranted. Many cases, however, are not so plain; none are in their early stages, when questions of prognosis and of treatment are of the utmost importance. Nevertheless, many cases seemingly latent may be recognized by careful

FIG. 9.



Area of dulness in aneurism of the descending portion of the arch of the aorta. The X shows the area of visible and palpable pulsation.

FIG. 10.



Area of dulness in aneurism of the ascending portion of the arch of the aorta. The X shows the area of visible and palpable pulsation.

inquiry into all the etiological factors present in the individual case, a minute study of all the clinical manifestations, and a painstaking physical exploration of the chest. Although we well understand that aneurisms are not extremely common, by which I mean to say that by far the majority of patients that come to us do not suffer with aneurism, it is well to bear in mind the possibility of the existence of aneurism in the event of certain unusual symptoms. The most important and suggestive of these are what are known as the pressure symptoms of aneurism,—the consequence of pressure exerted by the

aneurism on one or more of the structures contiguous to the arch of the aorta. These frequently are the first evidences of the existence of an aneurism, and their correct interpretation, the appreciation of their significance, often permits of the "happy guessing" of the presence of an aneurism long before demonstrable or dependable physical signs have developed. (Figs. 9 and 10.)

In the majority of cases the most noteworthy pressure symptoms are connected with the respiratory tract, and of these the most important are due to paralysis or spasm of one or both vocal cords, occasioned by pressure on the recurrent laryngeal nerve or nerves, generally the left. The most suggestive symptoms are a peculiar and quite characteristic cough, alterations of the voice, and dyspnoea. The cough of aneurism is brassy, metallic, clanging, or ringing in character,—a laryngeal cough; and though it may be, and usually is, forcible, it is somewhat lacking in the sharp explosive features of the "healthy" cough. It constitutes an extremely characteristic manifestation of aneurism of the aorta and of itself alone frequently permits of a presumptive diagnosis. The alterations in the voice have been well described by Walshe, who says that "the speaking voice may be husky, muffled, cracked, and hoarse, or simply weakened, or tremulous and variable in pitch, or actually lowered in register." In some cases complete aphonia results, but this is not usual. Laryngoscopic examination frequently reveals the larynx normal save in one respect,—the cadaveric position of the cord corresponding to the recurrent laryngeal nerve that is paralyzed. In these cases the other cord crosses the median line and renders phonation possible, but in consequence of unequal tension, unequal vibrations and hoarseness, or a peculiar monotone and inability to reach a high note, a falsetto voice, develops. Dyspnoea, which is often associated with marked stridor, that may be inspiratory or both inspiratory and expiratory, and which like the cough is often paroxysmal in nature, is an extremely characteristic symptom of many cases of aneurism. Sometimes the narrow glottis (occasioned by paralysis of the vocal cord or cords) is still sufficient for easy respiration during quiescence, but exertion produces increased inspiratory demand, and during the effort the paralyzed cords are violently drawn downward and inward with resulting apnoea. The cough and dyspnoea in these cases acquire an especial diagnostic significance, because they are laryngeal in type,—that is, they sug-

gest disorder of the abductors and adductors of the vocal cords, because the cough is disproportionate to, or exists independently of, catarrhal alterations of the respiratory tract demonstrable upon laryngeal or other examination, and because of the paroxysmal character of the dyspnoea which at one moment may be extreme and almost immediately entirely relieved.

In some cases such is the situation and the direction of pressure exerted by an aneurism that its existence may be inferred from some well marked alterations of the breath sounds such as amount to positive evidence that the access of air to the lung through one or the other division of the great bronchial tubes has been shut off or locally interfered with. In the majority of cases it is the left bronchial tube that is obstructed, and an impairment to a notable extent of the respiratory murmur of the left lung or of one of its lobes may, even in the absence of other physical signs, become a suggestive sign of a tumor compressing the lung itself or the bronchus leading to it. If the lung itself be compressed, the symptoms may not be very obtrusive, or they may indefinitely resemble those of pneumonia or pleuritis; but when the bronchus is compressed, dyspnoeic attacks of a highly paroxysmal character are quite frequent. Local diminution in the expansion of the lung is, of course, to be observed in these cases. If the pressure be long continued, retention of secretion, fetid bronchitis, and bronchiectasis may result. On the other hand, pressure on the bronchial arteries, occasioning defective nutritive supply, may lead to gangrene of the lung, or indirectly to pulmonary tuberculosis (a not uncommon complication of aneurism of the aorta).

Several symptoms intimately related with the trachea are of much value in the diagnosis of aneurism. Of these lateral (as well as anteroposterior) deviation of the trachea, which may be detected by examining the trachea in its course in the neck, and limitation of the normal upward and downward movement of the trachea during deglutition, though of value, are relatively unimportant when contrasted with the more common and more easily recognized sign known as tracheal tugging. This tracheal tugging, which is generally designated Oliver's sign, consists of a distinct tug or downward traction on the trachea occurring synchronously with each systole of the heart. A somewhat similar sign, known as Cardarelli's sign, consists of a lateral rather than a downward traction on the

trachea. Together, however, the two signs are frequently known as the Oliver-Cardarelli sign. The sign may be elicited by steadying the head of the patient with the left hand and with the thumb and index-finger of the right hand grasping the cricoid cartilage and gently pressing it upward. Should the sign be present, a distinct tug will be appreciated,—a tug that is distinct and not to be confounded with a slight pulsatile movement that may be felt even in some normal individuals. In some cases the sign may be elicited better by standing behind the patient, steadying his head against the body of the physician, and with both the index-fingers gently pressing upward the cricoid cartilage,—a method warmly advocated by Ewart, who has directed much attention to the sign. Though by no means pathognomonic, the sign is of much importance, being significant of an aneurism of the transverse or of the descending arch of the aorta, of an aneurism that is in relation with the left bronchus and the bifurcation of the trachea. And inasmuch as an aneurism that impinges on the bifurcation of the trachea and on one or the other bronchus, even when of considerable size, is frequently for a long time unattended with trustworthy physical signs, or is entirely devoid of physical signs, the diagnostic value of tracheal tugging may be very great. Rather recently Hall has described a tracheal diastolic shock which he regards as of importance in the diagnosis of aneurism of the aorta.

A number of other pressure symptoms are often of much diagnostic value. Thus the aneurism rather commonly is so situated as to interfere with the circulation of the blood in one arm or in one side of the neck and head. At times this results from direct pressure exerted by the aneurismal sac on the supplying artery; in other cases it results from the artery springing from the sac itself, the blood supply of the part in question being compromised by the blood having to pass through a larger or smaller sac more or less filled with blood-clot. Careful examination of corresponding arteries of the two sides of the body, the carotid, the subclavian, the axillary, the brachial, and the radial, in these cases will disclose differences in time and in force in the pulses; the one is retarded and weaker than the other,—a sign of much value in the diagnosis. Interpreting this phenomenon, however, one must bear in mind anatomical differences in the distribution of the arteries. Thus, I well remember a case, and by no means an isolated case, of a large sacculated aneurism

of the transverse and descending arch of the aorta interfering with the access of blood to the left carotid artery, the pulse of which was very small, while that of the right carotid was quite normal. The pulse in the left radial, though small, was much larger than the pulse in the right radial, which was almost imperceptible,—a phenomenon due to an anomalous distribution of the vessels in the right forearm, the pulse in the right brachial being much larger than that in the left brachial. As pointed out by Osler, a valuable sign in some cases of large saccular aneurism may be obliteration of the normal pulse in the abdominal aorta and its branches. At times, though scarcely as frequently as in intrathoracic tumor, obstruction to the venous circulation occurs. This manifests itself by marked congestion and swelling of the veins, and by cyanosis, and sometimes œdema, corresponding in situation and extent to the situation and the degree of the obstruction. In some cases more or less well marked evidences of a vicarious collateral circulation appear in the normally small veins beneath the skin. Clubbing of the finger-ends and incurving of the nails, usually unilateral and corresponding with the side towards which the pressure of the aneurismal sac is directed, have been observed in several cases.

Series of interesting phenomena induced by pressure on the ciliospinal branches of the sympathetic nerve are by no means uncommon. As might be expected, these are usually unilateral. Should the pressure be moderate, sufficient only to irritate or stimulate the vasodilator fibres, dilatation of the pupil results, and with this there is usually associated unilateral pallor of the face. Should the pressure be sufficient to paralyze or destroy the vasodilator fibres, the pupil becomes contracted and one side of the face hyperæmic and usually the seat of increased perspiration. Associated with the contraction of the pupil in some cases one may observe narrowing of the palpebral fissure and enophthalmos. In some cases of aneurism exophthalmos has been observed; in some cases it is attributable to venous engorgement, in other cases it is inexplicable. In quite a number of cases the detection of such apparently insignificant symptoms has led the observing physician to the recognizing of an entirely unsuspected aneurism.

Pressure upon the œsophagus is an important symptom in some cases; in other cases—cases of very large saccular aneurism—it appears remarkable that the œsophagus should, as it does, escape

implication. In the event of pressure, the subjective complaint, of course, is dysphagia; but dysphagia may be due also to pressure upon the vagus or its œsophageal branches. In the latter case there may be other manifestations of gastrointestinal disorder,—vomiting and diarrhœa,—but these are unusual. The dysphagia always suggests an œsophageal stricture, which in reality the condition amounts to. The important fact to bear in mind is that in case of suspected œsophageal stricture an œsophageal bougie or a stomach-tube should not be introduced until the possibility of an aneurism has been eliminated. On more than one occasion a stomach-tube has been unintentionally introduced into the sac of an aneurism with the immediate death of the patient. It is because of the possibility of the occurrence of such an unfortunate accident, and because I regard them as of little diagnostic value, that I have never been led to resort to œsophageal auscultation (of the murmur of an aneurism) by means of a solid œsophageal sound, or to the devices recommended by Schnell and by Porter, consisting of introducing into the œsophagus a blind soft rubber tube, which, having been filled with water, may reveal at its external end (to which a glass tube may have been attached) systolic and diastolic variations in the height of the water. In some cases pressure upon the thoracic duct is associated with the pressure upon the œsophagus. In the event of this combined pressure it may be difficult to determine to which the emaciation of the patient may be due, whether it is due to the pressure on the thoracic duct or to the pressure upon the œsophagus and the consequent ingestion of insufficient food.

Finally, a word with regard to the significance of pain,—a symptom that may be almost entirely absent in some cases, a symptom that in other cases is the most obtrusive manifestation of the disease. More likely to be present in deep-seated aneurisms, it is usually described as sharp and paroxysmal, or dull, boring, and persistent. In some cases pressure upon the intercostal nerves may give rise to the most intractable neuralgiform pains, which for a time may be the only noteworthy manifestation of the aneurism. Persistent pain beneath the sternum, or in the region of the fourth, fifth, and sixth thoracic vertebræ, is extremely significant. So impressed have I become with the importance of pain along the course of the intercostal nerves, or beneath the sternum, or in the region of the vertebras, that if it be persistent and unrelieved with medication, I

undertake a careful examination of the patient with a view to determine the presence of an aneurism. In several such cases I have thus detected an entirely unsuspected aneurism. In another case persistent and inexplicable paroxysmal shooting pains were present in both arms for several years, until finally they were correctly referred to an aneurism of the aorta. The association of an otherwise inexplicable cough, dyspnoea, and intrathoracic pain should always awaken the suspicion of deep-seated aneurism. A characteristic of the pain in some cases is that it is relievable by large doses of potassium iodide, a fact of some diagnostic importance. Some years ago Eichhorst drew attention to the fact that he had observed in three cases that percussion of the anterior chest over a localized area provoked severe pain and paroxysms of coughing, and that the cough was induced also by palpation of these areas. Upon these phenomena he based a provisional diagnosis of aneurism,—a diagnosis that was confirmed by necropsy in all three cases. He regarded the manifestations, therefore, as possessing considerable diagnostic importance.

To several other suggestive manifestations of aneurism, particularly to some well-known auscultatory signs, attention need not be directed here. Passing reference, however, may be made to the value of the X-rays in the diagnosis, and to an habitual premonitory bleeding that occurs in some cases, and that rarely is the first noteworthy symptom of the disease.

Inasmuch as many of the foregoing remarks are applicable alike to the diagnosis of aneurism and of solid tumors of the mediastinum, I should like now to direct attention to certain factors that may enable us to differentiate between aneurism and intrathoracic growth. Aneurism is most common in males, between the ages of thirty-five and forty-five years, especially in males who have been engaged in laborious occupations, who have become infected with syphilis, and who have used alcohol to excess. On the contrary, intrathoracic growths exhibit no special predilection for either sex, they occur at any age if secondary, but usually before the age of thirty-five years if primary, and occupation, alcohol, and syphilis (except in the case of gummas) are without etiological significance. A bulging, expansile tumor is, of course, pathognomonic of aneurism; in the case of intrathoracic tumor a primary growth elsewhere in the body or the development of a mass of enlarged glands above

one clavicle is likewise practically pathognomonic. In addition, œdema of one side of the chest, especially if unassociated with œdema of the corresponding arm, is extremely suggestive of tumor. A systolic or a double murmur, if unassociated with other evidence of valvular disease, is suggestive of aneurism; murmurs as such have no relation to intrathoracic growths, but they may develop if the heart be displaced or the blood-vessels be encroached upon or distorted. A palpable diastolic shock and a marked accentuation of the second aortic sound is quite common in aneurism, but absent in tumor. Tracheal tugging does not occur in tumor, though a slight movement (observable even in some healthy persons) may be detected. The effects of aneurism on the lung are usually those of compression only, whereas the effects of tumor growth may be manifest by consolidation, the growth invading the lung tissue and in some cases giving rise to metastatic nodules. In the case of tumor a clear, or probably more likely a blood-stained, fluid is common when the growth has reached and implicated the pleura; on the contrary, should an aneurism give rise to alterations of the pleura, acute pleuritis usually results, since the aneurism is more irritative than the more quietly progressing growth. Pain is usually less severe, and it is certainly less characteristic in tumor formation than it is in aneurism, though it may be entirely absent in some cases of aneurism. Pressure effects on the recurrent laryngeal nerve are much more suggestive of aneurism than of tumor. Under the beneficent influence of rest and other treatment the symptoms of aneurism usually, for a time at least, show amelioration, whereas almost without exception the symptoms of tumor are continuously progressive. The course of aneurism is very variable and the disorder may last for a number of years, whereas intrathoracic growths almost certainly result fatally within a year.

Neurology

SOME ASPECTS OF PARANOIA.

BY ROBERT H. CHASE, A.M., M.D.,
Superintendent of Friends Asylum, Frankford, Philadelphia.

THE general nature of paranoia is not very clear to the general practitioner, and even among asylum physicians the knowledge concerning it is vague, for few types of mental alienation have been the subjects of so much controversy and divergence of opinion. This critical analysis, going on for nearly a century, has covered a large array of pathological conditions, and has included many varieties of the affection, ranging from imbecility, on the one hand, to almost normal intellection on the other. In numerous instances the views of writers have been irreconcilable. This disease was first described in the beginning of the nineteenth century by Esquirol, under the title, *monomanie intellectuelle*. He designated it as "that form of insanity in which, while the memory, the conceptions, and judgments generally are not destroyed, and no pronounced emotional disturbance exists, yet the patient is controlled by some expansive delirium or ambitious project." He failed, notwithstanding his acumen, to grasp its essential nature, confounding it with other types of insanity not at all allied. The followers of this great teacher comprehended more fully its character, but in their hands the original term became overburdened with subdivisions of nomenclature, including so many shades of mental perversion that finally it lost its original use, and came to be applied only to certain special conditions or symptoms arising in any form of insanity. As a result, not a few English and American alienists of an early day denied it a place in their classifications. Its proper conception, however, continued to be recognized by French writers, to one of whom, Morel, belongs the credit of discovering its chief character in the persistence of systematized delusions founded on a neu-

ropathic basis. Among the Germans, Snell and Griesinger did much towards its elucidation, and to the former we owe the conception of its primary origin. One may obtain some idea of the perplexity that attended these early studies from the numerous synonyms that have been suggested in the past. Some of these terms are: *Monomanie intellectuelle* (Esquirol), *délire partiel*, *folie systématisé* (Morel), *primäre Verrücktheit* (Griesinger), partial insanity (Pritchard), chronic delusional insanity (Bucknill and Tuke), ideational insanity (Maudsley), *paranoia originaria degenerativa* (Morselli), monomania or monopsychosis (Clouston), delusional monomania (Spitzka), paranoia (Krafft-Ebing).

Classical paranoia is now defined as a definite symptom-complex, divided into three stages: (1) The stage of incubation, in which eccentricities, egotism, and suspiciousness play a prominent part; (2) the persecutory stage, marked by delusions of persecution and generally accompanied with hallucinations; and (3) the ambitious stage, characterized by change of the personality and by fixed and systematized delusions of grandeur. Some of the usual forms are described under the designations, persecutory, ambitious, religious, jealous, and querulous. All of these are varieties of the same inherent degeneration, and take their coloring from individual bias, accidental environment, or irregularities in its course. It is an intractable form of mental derangement that develops generally in persons with either acquired or inherited defects. It usually makes its appearance in the developmental epochs between fourteen and thirty-five years of age. The onset, in a large proportion of cases, is gradual and progressive; in others it is intermittent, going by fits and starts. Although it may apparently arise spontaneously, generally some slight exciting cause can be found, such as an acute illness, injury to the head, alcoholic excess, or so trivial a thing as the after-impression of a startling dream. Even the persistent harping on one string, as it were, in patients where the thoughts are constantly occupied with a real or fancied grievance, may be an important causative factor.

While the genesis of paranoia is usually very insidious and its early development tardy, it does not seem to be well understood that the active symptoms of the established disease may make their appearance abruptly, particularly so upon the occurrence of an exciting cause, such as mental stress or shock, business reverses, deprivation, or disappointment. The case of Michael Trimber is



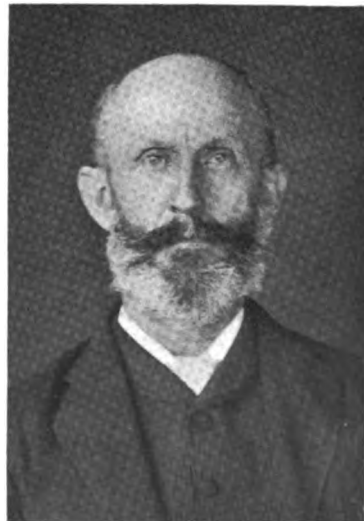
1



2

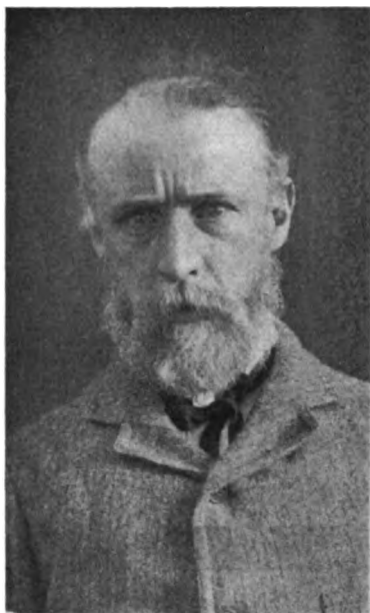


3



4

1.—Classical paranoia. 2.—Persecutory type. 3.—Homicidal type. 4.—Erotic type.



5



6



7



8

5.—Jealous type. 6.—Querulous type. 7.—Ambitious type. 8.—Religious type.

in point. This young man, while undergoing imprisonment for a lesser crime, stealthily killed his cell-mate without provocation. At the trial for murder, Dr. Ray, the celebrated alienist, testified to his soundness of mind under the belief that he was feigning insanity. Afterwards, Ray, having become convinced of his error, unequivocally acknowledged it, and the prisoner was acquitted on the ground of insanity. For more than ten years preceding his death he was a patient under the care of the writer, where his life in the hospital amply confirmed the verdict that placed him in the list of the criminal insane. The development of active symptoms in this case transpired within the short period between visits of his mother, who came to see him frequently in prison. One day, shortly before the murder, the mother said to one of the keepers as she was leaving the prison after her accustomed visit to her son, "I found my poor boy so changed to-day, I fear he is going crazy." This innocent remark of the mother had much weight at the trial.

The symptoms of paranoia may be many and varied, or they may be few and circumscribed. It should be remembered that abortive cases are not rare, and that the disease may be arrested in any of its stages. In some abortive cases an imperative conception, a morbid impulse, or a delusive suspicion or fear may be the only manifestation, and the subject show no other evidence of impaired mentality. *Folie du doute avec délire de toucher* is a form of abortive paranoia, as well as many of the morbid phobias, so familiar to the psychiatrist. The fundamental disorder in these cases is grave, and only requires, it may be, an adequate exciting cause to light into flame the inflammable fabric. This affection of which we speak is generally held to be a definite entity, that in the great majority of cases arises and runs an independent course; but, in like manner, as it may be arrested in its progress at any stage, so it may be complicated by other forms of insanity, such as mania or melancholia; and there are instances recorded in which general paresis has developed.

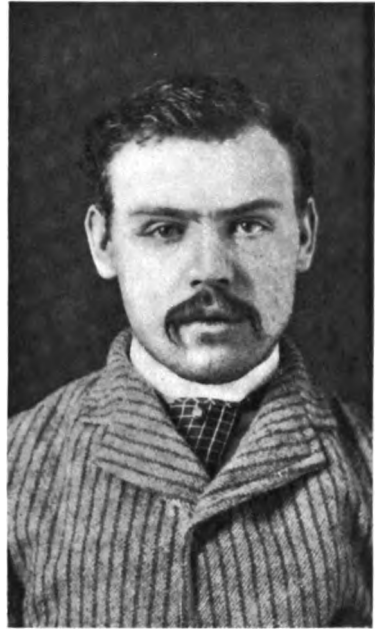
In medicolegal proceedings it is often difficult for the medical expert to satisfy his doubts that the prisoner is a paranoic, as was seen notoriously in the Guiteau trial, and in similar cases where the symptoms are masked by attempts on the part of the subjects to simulate insanity. A perplexing incident of this character occurred a few years ago at Moyamensing prison in the case of A. B., a subject of paranoia, who, by simulating the appearance of acute

mania, succeeded in deceiving a number of able specialists appointed to examine him. In expressing his opinion in court, one of the examiners, relying on his previous knowledge of the usually progressive and orderly development of the disease, could not bring his mind to the belief that prominent symptoms, however characteristic, would abruptly develop, even under the stress of imprisonment and by the shock of a horrible crime. The prisoner for many months successfully simulated a form of insanity different from the one with which he was afflicted under the tutorial influence of another prisoner, an adept malingerer, to the great distraction and annoyance of the prison community. Eventually he was shown in court to be insane, and was acquitted of homicide.

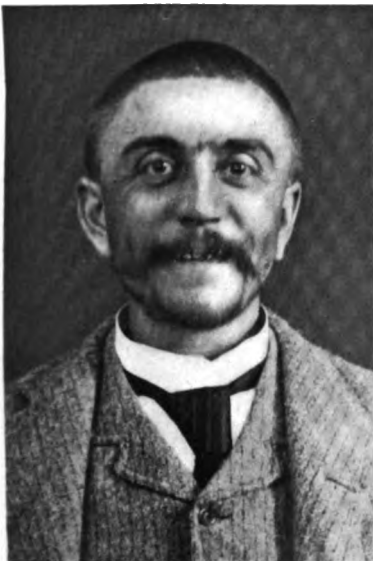
Numerically second only to classical paranoia is the primary or congenital form which was first outlined by Sander. In this group, as implied by the name, the fundamental defect dates in its inception from infancy, and is based on a degenerative constitution more pronounced than in the other types. The subjects of this class generally display marked mental and physical stigmata. The neural disorder in them is similar to that of idiocy and imbecility; some have epileptiform attacks, others choreiform movements, while hallucinations are not uncommon. To this class belong some of the noted assassins of history, such as Prendergast, although, on the contrary, many of the class are mild and inoffensive in character. An interesting case of the latter kind could be seen for many years at the Government Hospital for the Insane, after the war of the rebellion. He was a German by birth, dwarfish in stature, with a hydrocephalic head, and other stigmata that plainly marked him a degenerate. He had conspicuous hallucinations of hearing and delusions of an egotistical character. His self-esteem knew no bounds. Woe betide the visitor who incautiously expressed a wish to hear his verses, for there could be no escape on the lucky side of boredom. He would read in broken accents, with tobacco-stained mouth, from his effusions of no scant bulk, to the extent of some ten thousand hexameter lines. Besides filling the posts of commanders-in-chief of the opposing armies and navies in the war, he vaunted himself a great physician, healing human ills by the "rays of the eye," a virtue peculiar to himself. He had also hallucinations of smell, and he often complained of a gas that he called "acrimonialis," produced by unknown agencies.



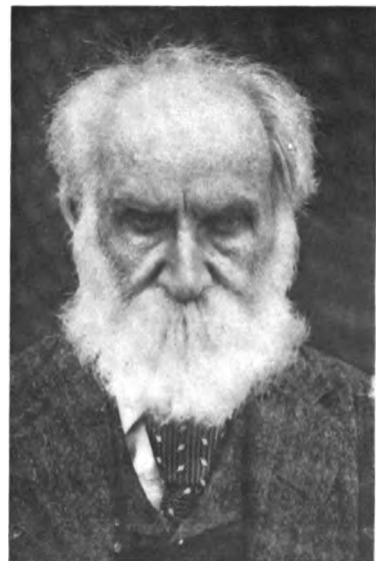
9



10



11



12

9.—Primary paranoia. 10.—Hebephrenia paranoïdes. 11.—Rudimentary type (folie du doute).
12.—Reasoning paranoia.

In a smaller group of cases Spitzka has proposed the name, *monomania sine delirio*, which other writers term reasoning paranoia. Pinel, the nephew, characterized these cases as "turbulent, indocile, quick to anger, committing outrageous acts, which they are always ready to justify by plausible reasons, and who are to their families, their kindred, and their friends continued subjects of anxiety and grief. They are continually doing wrong, either by neglect, by malice, or by wickedness. Incapable of mental or physical application, they destroy, subvert, and unsettle everything with which they are brought into contact and which they can injure." To this division belong many of the subjects that are classed by some authors under moral insanity.

Kraepelin describes a type of adolescent insanity as *dementia præcox paranoïdes*. It begins with restlessness, headache, emotional depression, passing rapidly into a delusional state with ideas of persecution. Soon or late in its course there may be megalomaniac delusions, often unsystematized. The disease speedily runs into confusional dementia. In the experience of many American psychiatrists this type has not been recognized, and it is probable that it is rare, at least in this country. In recent years the writer has been in the habit of ascribing the appellation, the paranoid form of adolescent insanity, or hebephrenia paranoïdes, to that group of patients that develop certain paranoid symptoms during the course of adolescent insanity. Reference here is not made to the classical symptoms of paranoia, but to a class that pursues a unique course, manifesting rather erratically many of the signs of primary paranoia. In some respects these manifestations are not unlike the symptoms of moral perversion in general paresis. Some of these cases exhibit a distinct tendency to crime, in others the bizarre conduct does not reach this extreme degree, but finds vent in oddities of behavior, either harmful or mischievous. It is probable that some of these instances would be classed by some authors as subjects of moral insanity. Of the authorities, Clouston recognizes a type of adolescent depravity which he differentiates from the common insanity of this period. The distinction that the writer would make is that while primary paranoia, like moral insanity, is based on imbecility, this type to which he refers does not so arise, but is an atypical variety of adolescent insanity.

TRAUMATIC LESIONS OF THE BRAIN IN THEIR RELATION TO OPERATION.

READ AT THE SECTION IN MEDICINE OF THE COLLEGE OF PHYSICIANS OF PHILADELPHIA AND REVISED FROM A LECTURE DELIVERED TO THE STUDENTS OF THE UNIVERSITY OF PENNSYLVANIA.

BY WILLIAM G. SPILLER, M.D.,

Assistant Clinical Professor of Nervous Diseases and Assistant Professor of Neuropathology in the University of Pennsylvania; Neurologist to the Philadelphia Hospital.

It is not my intention to discuss cases of bullet-wound of the brain or of head injury with fracture of the skull and depressed bone. Most surgeons, I think, would elevate a depressed piece of bone from the brain, especially if symptoms of compression were present; but in cases of fracture of the skull without displacement of bone it may be more difficult to decide whether operation is advisable or not. I wish to bring before you for consideration certain conditions in which the surgeon and the physician find much difficulty in deciding what is best to be done. Patients are sometimes brought to us in an unconscious state, and we may be unable to obtain any account of what has happened to them. An examination of the urine may reveal a large amount of albumin and granular casts, and we may be in doubt whether the case is one of uræmia, or uræmia associated with concussion of the brain, and possibly with alcoholic intoxication. A wound on the head is always suggestive of trauma of the skull or brain. I would urge that in every case of the kind referred to here the head should be shaved and a careful examination made for subcutaneous hemorrhage and fracture of bone.

We must remember that if a subdural hemorrhage is small the brain may accommodate itself to the altered conditions after a time, and the symptoms of compression may disappear, and yet a person with such a lesion of the brain is in danger of becoming epileptic or of developing traumatic insanity, it may be years after the accident has occurred. Only recently a young man came to

my clinic who had received a severe blow on the head and had been in a hospital but several days. Six or seven years later epileptic convulsions began and have persisted.

Many symptoms found directly after a blow has been received upon the head are referred to as shock, but some of these may seem to indicate a focal lesion which does not exist. A subdural hemorrhage may occur in a "silent" region of the brain and, possibly, by contrecoup hemiplegia and epileptiform convulsions may develop from the transitory disturbance of the motor cortex at a point opposite to where the blow was given. Too hasty operation may lead to exposure of the motor area when the persisting lesion may be in the occipito-parietal lobe of the opposite side. A case in which the symptoms were thus confusing is the following, the notes of which are from Dr. S. M. Hamill:

The patient, a man of middle age, was struck with a golf-ball just above and about an inch and a half posterior to the right ear. He fell on his face immediately. When his friends reached him, he was able to speak, and said he would be all right in a few moments. He sat up shortly and attempted to rise, but fell again. He was kept on the ground after that for ten or fifteen minutes; then with very little assistance he walked to a bench near by, where he rested for a time; then he walked to the club-house, about seventy-five yards away.

He was examined by Dr. Henry Norris at this time, and was then developing a slight headache, which soon became severe. He was placed on a sofa, and about an hour after the accident he began to vomit. After vomiting for a few minutes, he became unconscious and developed a violent clonic spasm, involving only the left side. Dr. Norris described this spasm as unusually violent. The right side was to no extent involved; his circulation became very much depressed; his pulse practically imperceptible at the wrist; his heart's action exceedingly irregular,—sometimes very slow and sometimes rapid and very weak. His respirations were irregular and snoring. Dr. Norris said that he fully believed the man would die in a few minutes. The spasm lasted for about half an hour. When it ceased, his right side was found paralyzed and he was entirely unconscious. His circulation improved under stimulation, and he was removed to his cottage three or four hours after the accident. His condition gradually improved during the night. By

the following morning he occasionally opened his eyes and began to move his right hand and forearm slightly.

I saw this patient on Saturday at midnight, in consultation with others, the accident having occurred the day before. The patient was unconscious and very paretic on the right side. We decided that an operation at that time would not be proper. There was no doubt that the golf-ball had struck in the right occipital region, and it was my belief that the hemiplegia would disappear within a few days and that it was caused by a bruising of the brain, or some similar condition, at a point opposite to where the ball had struck. The condition of paralysis improved persistently and rapidly, and had practically disappeared by Wednesday of the following week (the accident having occurred on the previous Friday). His lucid intervals became more frequent and prolonged up to Wednesday; from Wednesday until the following Saturday (the date of an operation) there was no pronounced change in this respect. Dr. Norris and Dr. Hamill were inclined to believe that this somnolence was more marked. He had retention of urine, and had to be catheterized up to the time of operation.

Dr. Hamill sent me a long telegram, explaining the patient's condition. This telegram, unfortunately, has been mislaid, but it gave me reason to believe that there was still increase of intracranial pressure, and therefore I recommended operation at once at the spot where the ball had struck. I believe this advice was also given by Dr. Joseph Collins and Dr. Graeme M. Hammond, who were called in consultation.

Dr. Bull, of New York, trephined the skull immediately over the point of injury; there was no fracture. Immediately underlying the dura was a firm jelly-clot, about the size of the palm of one's hand, which was readily removed. The patient reacted well from the operation. His lucid intervals rapidly became more prolonged, and within three or four days he was easily roused and rational practically all the time. Within two or three weeks he was able to be up and about, and from that date his convalescence was rapid.

He has been in better health since the operation than before. If there has been any change in his disposition at all, it is that he is less irritable than before his illness. He had some paralysis of the muscles of the right eye; but it is impossible to say now

which ones were involved. There was some weakness of these muscles for several months after the operation, and the patient held his head elevated and turned a little to the right in endeavoring to focus upon objects. This disappeared entirely. Dr. W. C. Posey's eye examination, made prior to the operation, revealed nothing of interest.

In this case and in the one following external ocular muscle paralysis was noticed with a subdural clot in the occipital region. It is important to remember that such a paralysis does not necessarily imply that the lesion is at the base of the brain.

I give briefly the notes of another very similar case:

D. McD. was admitted to my service in the Philadelphia Hospital, September 18, 1902. He was said to have had convulsions of the right upper and lower limbs, with vomiting, two days previously, after a protracted debauch, and to have fallen and struck his head. He was not epileptic.

He was admitted to the hospital in a semiconscious condition. He could not answer questions, but moaned, sang, or talked irrationally, at intervals. The upper limbs were held rigidly across the chest, and the lower limbs were also very rigid. At times he had convulsive movements of the left side of the face without implication of the rest of the body. When his head was shaved a large contusion was found on the left side, behind the ear, in the occipital region, and he had bruises all over the body. The pulse was slow and weak, the pupils were dilated, the arteries were sclerotic, the respiration was slow, deep, and full, the heart-sounds were normal. The patellar reflex was diminished on each side. He was seen by the resident physician and the nurses in several convulsive attacks of the left side of the face, the right side of the body not being implicated. The urinary examination on admission showed a specific gravity of 1020, a heavy white sediment, a large amount of albumin, and numerous granular and hyaline casts.

The convulsive attacks upon one side of the face still continued, and it is probable that occasionally they were on the right side. The temperature at first was 101° F., but it soon became normal. On September 28 the stupor became more marked, and continued during the entire morning. At 2 P.M. of this day he had a convulsion, which was seen by the resident physician, and was confined to the right side of the face. The muscles of the forehead

were not implicated. The first finger of the right hand twitched, but there was no movement of the right lower limb. An examination made this day, after the patient had been treated vigorously for uræmia for ten days, showed no albumin and only one or two hyaline casts. The tendon reflexes of the upper limbs were not exaggerated, while those of the lower limbs were diminished on each side. The Babinski reflex was distinct on each side, the big toe being moved upward. The subcutaneous hemorrhage in the left occipital region had almost disappeared, but considerable thickening of the soft tissues in this part remained. When the man was stuck with a pin, he placed his hand on the irritated spot, showing that he was not paralyzed. It was impossible to determine in any way whether hemianopia was present or not. The presence of right lateral hemianopia, such as would be caused by pressure from a clot on the left occipital lobe, would have been of aid in deciding regarding operation, but it was impossible to test for this even with the feeding-cup, because of the stupor. Pressure about two inches above the left ear caused pain. The right pupil was slightly larger than the left, and the muscles on the left side of the face possibly were a little better innervated than those on the right when the face was irritated by a pin. There was weakness of the left external rectus muscle.

I decided that no further delay in operation would be permissible, and Dr. A. C. Wood was called in consultation. Dr. Wood agreed that an operation was advisable, and trephined directly at the spot where the subcutaneous hemorrhage had been observed behind the left ear, and the opening was enlarged by rongeur forceps. The skull when exposed was found intact. The dura bulged into the trephine opening and the brain did not pulsate. When the dura was incised, a blood-clot was revealed directly at the seat of operation. It was about the size of the palm of the hand, and was removed, considerable blood-stained fluid escaping. After the operation there was some delirium and some protrusion of the eyeballs, but on the second day after the operation the patient replied correctly to questions, which he had not done previously during the time he was in the hospital. He made a complete recovery, although he still has some mental confusion, which in a person who has been so profoundly alcoholic is not surprising.

In some cases operation for the relief of pressure is so impera-

tive that a delay of a day may be fatal. This is illustrated by the following case:

F. S., twenty-eight years old, had been found lying by a railroad track. He was admitted to the Polyclinic Hospital, February 22, 1902, to the service of Dr. John B. Roberts, in an unconscious condition. The scalp over the left parietal eminence was lacerated. The wound was probed, and as no fracture was found, the skin and subcutaneous tissues were sutured. The right pupil was dilated more than the left. He developed bilateral epileptiform convulsions. He was catheterized, and the urine was found to contain albumin and granular casts.

At 7.30 P.M. of the same day Dr. Roberts made an incision in the lacerated area of the scalp and found the skull normal. Much blood escaped during this operation. The patient was cupped over the kidneys and given a pilocarpine sweat. He had a convulsion in the operating-room immediately after the operation, and had eighteen convulsions in rapid succession until 7 A.M. the following day. These convulsions began with nystagmus and twitching of the eyelids. Both eyeballs turned to the extreme left. The left side of the face twitched and the mouth was drawn far to the left. The left upper limb became rigid and shook a little. Finally, the right upper limb twitched slightly, and, as the convulsion was ending, the right upper limb was invariably raised towards the head. The convulsions stopped abruptly at 7 A.M., February 23, but began again at 10 A.M., February 24, since which time there were a few resembling the convulsions described above. The left arm appeared weak, and the patient could not be made to use it. The eye-grounds examined by Dr. Schneideman were found normal.

I saw the patient on the afternoon of February 24, and because of the stupor, and the left-sided paresis, and convulsions confined to the left side and developing *after* the general convulsions had disappeared, I believed we had to deal with focal symptoms, and I recommended immediate operation at the right motor cortical area. For certain reasons it was decided to postpone the operation until the next day.

The convulsions ceased at 6 P.M., February 24. At 8 P.M. a marked change occurred. The man became much weaker and breathed laboriously. For a time he frothed at the mouth.

One pupil was greatly contracted, the other moderately dilated. Both sides of the face were equally relaxed. Both upper limbs were somewhat rigid and equally so, but the patient still moved only the right hand to the head when the supra-orbital nerve was pressed upon. The temperature was 104° F. and respiration 32. The man died February 25, at 1.20 A.M.

A necropsy made by Dr. Wadsworth revealed a large subdural clot extending over the right motor region and a hemorrhage in the right frontal lobe and one in the right occipital lobe. The skull was not fractured.

I give briefly the notes of the following case in which operation was determined upon and then abandoned, because of the improvement in the patient's condition. The man who committed the assault was in prison, and would have been held for murder if the patient had died. This to some extent complicated the case.

M. McL. was brought into the Polyclinic Hospital, in Dr. M. J. Stern's service, September 20, 1902, with an incised wound of the left cheek below the eye and under the effects of alcohol. He was believed to have fallen while in a fight. The pupils were equal. There were no signs of paralysis, and no bleeding from the nose, mouth, ears, or eyes. He had a slight contusion in the right occipital region posterior to the mastoid process. The pulse was of fair volume and rate and was regular. The man could be aroused with difficulty and was in marked stupor.

On September 21, 1902, the patient voided his urine and his bowels moved. His condition remained about the same, except that he showed signs of beginning restlessness and had vomited two or three times. He did not like to be disturbed. The pupils were equal and the irides reacted to light. The tongue was protruded straight. The patellar reflex was exaggerated and ankle clonus was present. The temperature was 100° F.

On September 29 his condition remained about the same, except that he had two epileptiform convulsions. These were not observed when they began, but when seen were general, and lasted probably about a minute. He answered questions incoherently and probably did not recognize any one. It was difficult to make him take any nourishment. The urine contained no albumin and no sugar.

The man was seen by me on October 1, 1902, and my notes made at that date are as follows:

"His stupor is so great that he makes no attempt to take the feeding-cup in his lips when brought from either side. His speech is distinct. According to the nurse's statement he swallows without difficulty. When his face is stuck with a pin he will not draw the corner of the mouth up on either side, but this may be on account of the stupor. The iris contracts promptly to light on the left side, but not so promptly on the right side. The right pupil is a little larger than the left. The movements of the eyeballs seem to be free in all directions. The patient moves all his limbs freely. The tendon reflexes of the upper limbs are not very prompt. The patellar reflex is very prompt on each side. There is no ankle clonus and no Babinski reflex. The prick of a pin is perceived on each side."

An examination of the urine, made October 11, 1902, showed albumin and abundance of pus cells, but no casts.

An operation was determined upon, as until October 7 very little improvement in the patient's condition had been noticed. Improvement became quite marked on October 8 and 9, and the operation was abandoned.

Although this man has made a recovery without operation, I feel that he is in some danger of becoming epileptic or of developing traumatic insanity. For nearly three weeks after he had received a head injury he was semiconscious.

In contrast to my first and second cases, in which the lesion of the brain was at the part where the blow was received, I give the notes of the following case to show that the effect of a blow may be exerted directly opposite the point of impact.

R., about fifty-four years of age, on April 8, 1902, fell from a car and struck on the top of his head. He had no signs of fracture of the skull on inspection or palpation. He was unconscious for fifteen or twenty minutes, but was not paralyzed in the limbs. He got up and walked a short distance. He was seen by me in the University Hospital, May 6, 1902. He was entirely unable to swallow solids or fluids, he spoke in a whisper, and had complete paralysis of the left vocal cord, and the left soft palate was not quite as well innervated as the right. The eye-ground could be examined only in the right eye, and was normal. I have already lectured on this case to the students of the University of Pennsylvania as one of fracture at the base of the skull, or possibly of

hemorrhage at the base without fracture. Operation was considered impracticable. The necropsy revealed a fracture passing through the left jugular foramen and causing complete degeneration of the left glossopharyngeus and vagus nerves.

And, finally, one case to show that a depressed fracture of the skull may escape detection after careful examination, and that by such a symptom as word-deafness we may determine the point of operation.

W. J. was admitted to the Polyclinic Hospital, May 12, 1901, to the service of Dr. Steinbach, about noon. He had been struck in the left temporal region with a baseball bat. On admission he had profuse hemorrhage from the left ear, which continued most of the night following. He had been unconscious since admission, and had tried to get out of bed, and did not seem to understand when spoken to. He took only a small amount of liquid food. The pulse was 60 and full, the temperature 100.2° F., the respiration 14. The hemorrhage from the ear ceased entirely, and he remained in a semi-comatose state most of the time.

On May 15 he was still in an unconscious condition and was somewhat restless. There was no conjunctival hemorrhage. The pulse was a little better. His bowels were kept open by enemata.

On May 17 paralysis both motor and sensory appeared on the right side. The pulse was slow and more feeble than before. He was still unconscious.

On May 20 the paralysis in the right lower limb seemed to be disappearing, and he understood some things said to him. He tried to answer questions, but could not make himself understood.

On May 24 he moved his right upper limb. He had a twitching of the right side of the mouth, but he also had a scar from a burn over the right facial nerve.

He was seen by me on June 6, 1901, and my notes made at that time are as follows:

"The convulsive tic of the right side of the face in its lower portion is still present. The paralysis on the right side of the body has almost entirely disappeared. His speech was as follows: 'Well sir I know I use to have good I can ever do ever know yes I been plenty I know where I at who I not I would to work to work plenty plenty.' This paraphasia was mingled with unintelligible words. This was his reply to the question of his name. He did not recognize his name when it was mentioned with other

names. When he was told to sit up in bed, he did so; when told to lie down in bed, he did so. When he was told to give me his hand, he did not seem to understand. He sat up in bed a second time on command, when all gestures were avoided. He seemed to have a slight restoration of word-hearing."

Because the right hemiplegia, which developed five days after the head injury had been received, had disappeared first in the lower limb and then in the upper, I believed an intracranial hemorrhage had occurred, and that the increase of pressure was from below upward. As the blood was partly absorbed and the clot retracted, the pressure would be removed first from the upper motor centre,—that of the lower limb,—and paralysis would first disappear in the lower limb. The persistence of word-deafness showed that the lesion was in the posterior part of the first temporal convolution of the left side, and this area was selected as the seat of operation.

On June 15, 1901, Dr. Steinbach made a horse-shoe shaped incision with the base downward over the temporal region, and exposed a stellate fracture. At the superior border of the squamous portion of the temporal bone was a depressed piece of bone. The skull was trephined just above the area of depression, and with the rongeur forceps an opening was made about the size of half a dollar. The dura was found torn and the brain substance protruding. The finger on being inserted entered a large cavity filled with degenerated brain tissue and blood-clots. These were removed and the cavity irrigated with normal salt solution. The wound healed nicely. The patient made a complete recovery, and his word-deafness in large measure passed away gradually, but about a year after his injury hallucinations of hearing and sight and delusions of persecution developed.

From the recital of these illustrative cases it will be understood that the question of operation in cases of injury of the brain may be very important. I think it is wiser in a case of very grave doubt to operate than to run the risk of death from a persistence of increased intracranial pressure, or the risk of future traumatic insanity or epilepsy. We cannot always prevent those mental conditions, even by operation, but we may lessen the danger of their development. Every case must be the subject of special study, and it is impossible to lay down hard and fast rules.

THE DIAGNOSIS OF FUNCTIONAL AND ORGANIC HEMIPLEGIA.

A CLINICAL LECTURE DELIVERED AT THE WESTMINSTER HOSPITAL.

BY PURVES STEWART, M.D., M.R.C.P.,

Assistant Physician to the Westminster Hospital, London.

GENTLEMEN,—When one gives demonstration of neurological cases to a post-graduate audience there is a temptation to select as one's subject some uncommon form of nervous disease. But although we have in the hospital at the present moment a considerable number of rare cases, I have thought it better to take up some subject of every-day practical importance rather than to indulge in a mere display of what might be termed neurological gymnastics, and therefore I propose that we should consider to-day some points in the diagnosis between functional and organic hemiplegia.

The first case that I show to you is that of Catherine W., a married woman, forty-eight years of age, who exemplifies very typically the ordinary clinical picture of an old hemiplegia. Her previous health has been good, on the whole. She has had neither cardiac nor renal disease. She has had nine children and two miscarriages. She was quite well, so far as she knew, until five years ago, and then one day, when she was feeling particularly tired and "done up," she felt giddy, gradually lost consciousness, and remained unconscious for several days. When she recovered consciousness, she found that her speech was indistinct, that her face was drawn to one side, and that she had lost all power in the left arm and the left leg. The speech returned in a few days, the left leg gradually improved to some extent, but the left arm has remained persistently paralyzed.

Let us examine her present condition together. You will observe that she is a somewhat stout woman, and when we talk to her we find that she is quite intelligent and able to answer questions. Her speech and articulation are normal. Now as to the rest of her

nervous system. First of all, in a case of hemiplegia we should never forget to examine the visual fields. (To the patient:) Look at me and tell me when you catch sight of anything moving. (Tests vision.) This is a good way of testing a patient's fields of vision. It is not necessary to buy a twenty-guinea perimeter in order to detect hemianopia. Stand opposite the patient as I do now. To examine her *left* visual field I make her cover up her right eye, meanwhile I close my left eye and gaze into her left eye with my own *right*. Then, holding a small piece of white paper at the end of a black pen-holder, midway between the patient and myself, I gradually bring it inward from the periphery, first from one side, then from the other. If her field of vision is normal, she and I ought both to catch sight of the white moving object at the same time (presuming that my own field of vision is normal). We do this with each of her eyes separately and find that her visual fields are normal.

When we look at her pupils we observe that they are not quite circular in outline and that the right pupil is not exactly in the centre of the iris. But they contract well when we bring a light in front of them and also when she converges to look at my finger. The pupils, then, are irregular, but they react normally. The external ocular movements are normal. The optic disks are healthy.

She has no anæsthesia of the face, trunk, or limbs. You observe that at rest her face is symmetrical, but when she shows her teeth the left nasolabial fold is not so deep as the right. The upper face is practically symmetrical on movement. The tongue comes out straight. With regard to the left upper limb, you will notice that it is rigid at all joints, that it is adducted at the shoulder, semi-flexed at the elbow, the hand pronated, and the fingers rigidly flexed over the thumb, which is turned inward. You see that she can make only a very feeble movement of the shoulder; she cannot move the elbow; she cannot move the wrist or fingers. She tells us, however, that sometimes the paralyzed fingers become unflexed for a moment and spread out when she yawns. So much for the upper extremity.

The lower extremity is also very severely paralyzed. It is rigid and in the typical posture of hemiplegia,—slightly flexed and adducted at the hip, extended at the knee and ankle. She cannot walk well; she drags the left foot along the ground. On testing the movements of the lower extremity we find she can feebly move the

hip and knee, but she has no movement in the ankle or toes. That is an important point. Then as to her reflexes. In the left upper extremity I tap the styloid process of the radius, and you observe how the supinator longus at once starts out. On the right side this jerk is much less lively. This patient also shows a reflex which was described by Bechterew, of St. Petersburg, last year, as the "scapulo-humeral" reflex. It consists in this, that if you tap the vertebral border of the scapula near its lower end, in a normal case you get practically no movement, but in a case of hemiplegia such as this you get a movement of the deltoid, sometimes also of the infraspinatus and teres minor. In the left lower extremity the reflexes are also much exaggerated, the knee-jerk is increased, and there is ankle clonus. On the right side this patient shows the normal flexor type of plantar reflex, on the left we have the abnormal extensor response.

As to diagnosis, in every case of paralysis we have first of all to ask the question, Is there an organic lesion present? In a case like this there is no difficulty in saying that there must be an organic lesion. The patient's history, the posture of the paralyzed limbs, the type of paralysis, the reflexes, etc., are all characteristic of a hemiplegia of organic origin.

Having decided, then, that our case is due to an organic lesion, we must ask ourselves two more questions. Firstly, Where is the lesion? and, secondly, What kind of lesion is it? First, Where is the lesion? You can generally answer that question by a study of the patient's symptoms. What is the lesion? You answer that usually by a study of the history. Now, as to where the lesion is. It is perfectly obvious that the paralysis of the face, arm, and leg on the left side must be referred to some lesion in the right side of the brain. The absence of hemianopia and of hemianæsthesia shows that the lesion must be one affecting simply the motor path. It is known that the fibres from the motor cortex go down through the internal capsule and then run down to decussate in the bulb. The patient has a lesion in the right side of the cerebrum. This might be either a large lesion in the cortex or, what is much more conceivable, it is a smaller lesion deeper in, in the region of the internal capsule, but not far enough back to catch either the visual or sensory paths.

The second question is, What is the nature of the lesion? The history in this case is that of a sudden onset. And you may take it

as a general rule that a sudden onset means a vascular lesion. That might be either embolism, hemorrhage (that is to say, rupture of an artery), or it might be thrombosis. Which is it in this case? Is it embolism? There is no source for an embolus here; the patient's heart is healthy and she has no signs of valvular disease. Is it hemorrhage or thrombosis? This is sometimes a very difficult question, and in many cases it is almost impossible to decide. You may take it as a general rule, however, that if the arterial pressure at the time of the paralysis was *plus*, it is more likely to be a hemorrhage than a thrombosis. For example, if a patient has chronic renal disease with cardiac hypertrophy and arteriosclerosis, it is likely that such a patient would have hemorrhage rather than thrombosis. Again, if a patient has miliary aneurisms in the cerebral vessels, that patient also is very liable to have a hemorrhage. Now, this patient shows no signs of arterial degeneration or of chronic renal disease. What about thrombosis? You may also take it as a general rule that in cases where the arterial pressure was *minus* at the time of onset, it is more likely to be a case of thrombosis. The commonest history of such cases is that the patient wakes up in the morning, and when he tries to get out of bed he finds himself paralyzed on one side. You must not imagine that the patient became paralyzed at that actual moment. The chances are that it came on during sleep. In this woman the paralysis came on at a time when she was feeling tired and "played out." But there is one further question about which many of you, I know, will be thinking. You will say, "But thrombosis does not occur in an artery if it is healthy." Thrombosis practically only occurs in a diseased artery. And in what diseases of the arteries are we most likely to have thrombosis? Firstly, you may have thrombosis in an atheromatous vessel in senile cases. But this patient shows no signs of the atheroma of old age. What is the other cause of arterial disease and in comparatively early life? It is syphilis. Now, are there any signs in this case to lead us to suspect a syphilitic endarteritis? Yes, there are. Remember that this patient had a couple of mis-carriages. That of itself might not count for much, but when we take with it the irregularity of the pupils and the fact that one of them is not quite central, these are strongly suggestive of old specific disease. So that the diagnosis we come to is that there has been a thrombosis of one of the arteries in the region of the right internal capsule.

The second case which I show you is that of a little girl, Elizabeth J., seven years of age. She was quite well until she was two years old, and then she had what her mother calls a "fit." She became drowsy and listless, and at the end of four or five days it was observed that she could no longer move her left arm or leg properly. Since then she has always been weak in that arm and leg, and has also had a number of fits in which the left arm and leg jerked. Now, when you look at this little child you see that both at rest and on movement the face is practically symmetrical. Her optic disks and visual fields are normal; so are the pupils and external ocular muscles. She has no anaesthesia of the face, trunk, or limbs. When you look at the left upper extremity you see it is constantly undergoing slow, involuntary movements at all the joints, especially at the fingers, but also at the elbow and shoulder. The patient can also move the limb voluntarily at all joints, but it is somewhat rigid and distinctly weaker than on the right side, and this weakness is most evident at the periphery of the limb. The lower extremity is also characteristic. The hip is slightly flexed and adducted, the knee is extended, and the heel does not quite reach to the ground. (Fig. 1.) The entire limb is somewhat rigid, but she walks fairly well. Now with regard to her reflexes. The wrist-jerks on the left side are much brisker than on the right. There is no ankle clonus, but here again we find the plantar reflex is of the extensor type on the left side and of the normal flexor type on the right.

What is the diagnosis here? There is no doubt that we have to deal with a case of organic hemiplegia. And where is the lesion? The paralysis of the left arm and leg shows that the lesion must be on the right side of the brain. The conjunction of localized spasms with paralysis points to the cerebral cortex, and this is the common site of lesion in children. What is the nature of the lesion? Some pathologists hold that these infantile cerebral palsies are due to thrombosis of cortical vessels, others hold that they are due to encephalitis or inflammation of the brain-matter, and that the thrombosis is only a part of such encephalitis.

The third patient whom I will show you is this young woman in bed, Johanna H., twenty-three years of age. She is a married woman, and was sent here because she had paralysis of the left arm and leg and was unable to speak. Her history is interesting. The family history is quite unimportant. She herself has never been

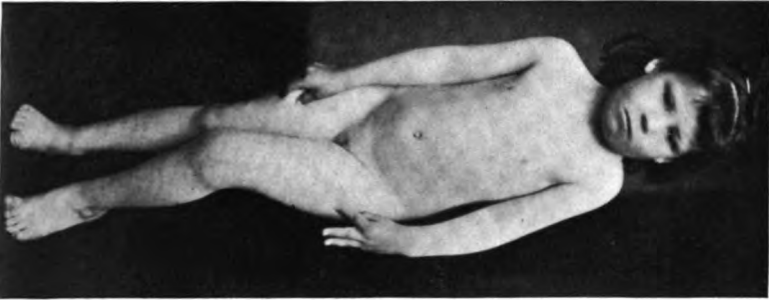


FIG. 1.—Old infantile hemiplegia.



FIG. 4.—Hysterical hemiplegia, showing gloriohysterical spasm of left side.

strong and is naturally somewhat nervous. She has been married three years. Two years ago, ten days after the birth of her first child, she had a fright,—her bed suddenly caught fire. She thereupon took a “fit,” lasting for six hours. Of this we have no description, but for two days after the fit she could not speak. She recovered completely. Then, seven months ago, three weeks after the birth of her second child, she had another “fit,” without any apparent exciting cause, in which she fell down, became unconscious, had twitching of the mouth, and sudden “jumps” of the legs. After this convulsion she was speechless for a fortnight, and it was noticed that the left arm was somewhat weak for a couple of months. She got over that, and three weeks ago, when she was sitting in a chair, she suddenly felt giddy and had a third “fit.” She fell down, had stiffness of the legs, threw herself about, and was unconscious for half an hour. After that she remained speechless for some days. On the third day of her illness she was admitted here, quite speechless, and with paralysis of the left arm and unable to stand. Five days from the onset of this attack she began to talk in whispers, but her left arm has remained stiff.

Her present condition is interesting. You observe that she looks pale and languid, but she is perfectly intelligent and her speech and articulation are normal. Her optic disks are healthy. When we examine her visual fields we find that they are concentrically contracted. This contraction is much more marked in the left eye than in the right. I show you charts of her visual fields which we have made with the perimeter. (Fig. 2.) Observe also that she can smell cloves and vinegar on the right side, not on the left. Similarly, she tastes quinine and sugar on the right side only. We test her hearing, and we find she can hear the ticking of a watch at twelve inches in the right, at six inches in the left ear, and when we try with a tuning-fork on the vertex we find that there is a similar diminution of hearing in the left ear.

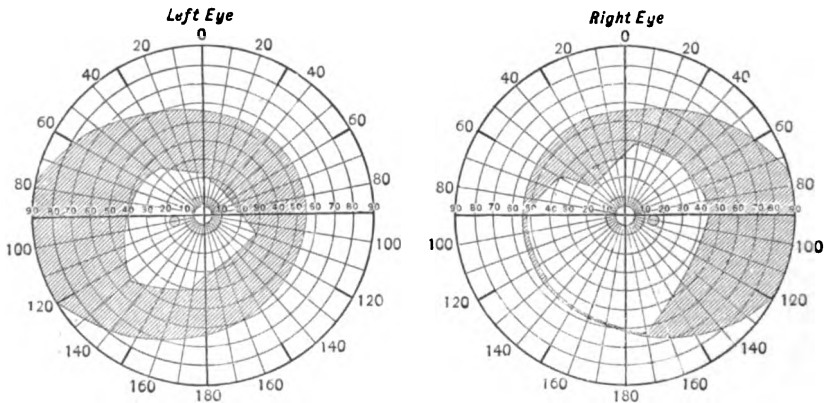
On examining her sensibility to touch and to pin-pricks we discover that she has areas of anæsthesia and analgesia on the left side of the tongue, on the left lower face, and all over the left upper limb, as you can see by the accompanying chart. (Fig. 3.)

Her pupils and external ocular movements are normal. The face, even at rest, is slightly tremulous about the lips. When she puts her tongue out it deviates to the left side, and you see that the nasolabial fold at the left side of the mouth becomes exaggerated.

This is a somewhat rare condition known as "*glossolabial hemispasm*." I have a photograph of her face which I will show you. (Fig. 4.)

When we examine her other motor functions we notice that the left upper extremity is rigid,—rigid at the shoulder, elbow, and wrist. But observe particularly that the fingers are quite flaccid. There is no absolute paralysis of movement at any joint, though all movements of the limb are excessively feeble. With regard to the lower extremities, there is marked weakness of the left leg at all joints, but here again no movement is absolutely lost. We now get her up to walk with the help of two nurses, and at once we see that her gait is quite different from that of the hemiplegic we saw

FIG. 2.



Crossed amblyopia.

first. Observe how she drags the back of the left foot along the ground; you see she drags the dorsum instead of the sole. A gait similar to that we can diagnose at once; it is a functional gait. Her wrist-jerks are equal; there is no ankle clonus, and you also see that when I scratch the soles of the feet I get flexion in each case.

To sum up this patient's case: She has now had three "fits," each followed by loss of speech. The first time she simply had a "fit," followed by loss of speech; the second time, in addition to the loss of speech there was slight weakness of the left arm; the third time (the present attack) she had the same thing with, in addition, stillness and weakness of the left arm and weakness of the left leg. She now has glossolabial hemispasm on the left side, weakness of the left arm and leg, loss of smell and taste, diminution of vision and

of hearing,—all on the left side,—and areas of anæsthesia of the left tongue, left lower face, and left upper extremity.

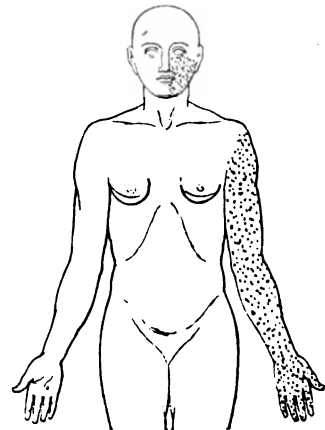
These are the facts. And here again we have to ask, Is this a case of structural, organic disease, or is it a case of so-called “functional” disease?

I do not propose—time does not permit—that we should discuss the very wide and very interesting problem as to the real nature of what is called functional disease. Let us be content to say just now that we apply the term “functional”—and “functional” is a better word than “hysterical,” which refers to a supposed connection with the uterus—to those cases in which we can detect no pathological changes by the most careful microscopic methods,—cases, in fact, which have no recognizable morbid anatomy.

But to return to our patient. Is this a case of organic or of functional disease? At first sight, the loss of speech and the paralysis of the limbs on one side might suggest that this was a case of hemiplegia with aphasia. In fact, the patient was actually sent into the hospital with such a diagnosis. But you will notice that the hemiplegia is on the wrong side. Cases of organic hemiplegia with aphasia in right-handed persons are on the right side. This patient is right-handed. Again, this patient's affection of speech, as we have observed it, has not been a true aphasia; it was simply a case of dumbness, or mutism, followed by a stage in which she whispered in monosyllables, and that was succeeded again by a stage in which she recovered the whole of her powers of speech. This, then, is not a case of ordinary hemiplegia with aphasia.

Moreover, she shows either diminution or loss of all the special senses on the left side. That is a combination which never occurs in organic disease; it is a symptom practically pathognomonic of functional disease. What about the posture of the patient's arm? That is suggestive at first sight of an old organic hemiplegia with contracture. But notice that although she is as rigid as possible at the shoulder, elbow, and wrist, her fingers are perfectly flaccid.

FIG. 3.



Hysterical hemiplegia; areas of anæsthesia on paralyzed side.

That is the converse of what you get in organic cases. Lastly, the patient's reflexes are against a diagnosis of organic disease, and in particular the plantar reflexes are both of the normal flexor type.

This, then, is a case of functional or hysterical paralysis. Hysteria has been called "*la grande simulatrice*," or the champion imitator. But we must remember that hysteria is not synonymous with shamming or malingering. This patient, for example, probably never saw a case of hemiplegia in her life. Hysteria is a real disease, as real as smallpox or appendicitis. The involuntary simulation of hysteria is always incomplete, and hysteria makes two mistakes in its imitations of organic disease: firstly, it makes errors of omission; it "leaves undone those things which it ought to have done,"—certain signs of organic disease are absent,—and, secondly, it makes errors of commission; it "does those things which it ought not to have done,"—it has certain additional signs which do not occur in organic disease.

Let us briefly recapitulate the points, as exemplified in this case, in which functional hemiplegia differs from organic. Firstly, as to the sensory symptoms. This patient has several areas of anæsthesia on the paralyzed side. These might conceivably be compatible with an organic hemiplegia. But observe that the anæsthesia is characteristically segmental in distribution. In the upper limb it stops short at the shoulder-joint, in the face it is strictly limited to the infraorbital region, and in the tongue it affects exactly one-half—in fact, all those parts which are in a state of spasm. This peculiar distribution of anæsthesia does not occur in organic hemiplegia. Secondly, with reference to the special senses. As we have already mentioned, loss of all the special senses on the paralyzed side does not occur in organic hemiplegia. Moreover, the affection of the visual fields in particular is quite different from what we get in organic cases. In an organic hemiplegia, if there is any affection of the visual fields from interruption of the visual path, it is usually in the form of a hemianopia; that is to say, the right half or the left half, as the case may be, is lost in both eyes, the patient is blind on his paralyzed side. But in hysteria the affection of the visual fields is not a hemianopia, it is a concentric diminution of the field of vision. Half is not wiped out, the field is simply restricted all the way round, and that diminution is more marked in the eye of the paralyzed side. The field is also slightly diminished on the non-paralyzed side. This is called "*crossed amblyopia*."

Then with regard to the motor functions. This patient shows what is called glossolabial hemispasm, which is by no means a common sign, but when it occurs it is practically pathognomonic. When the patient puts her tongue out there is deviation of the tongue and spasm of the face. You observe that the tongue is pushed out towards the left side. That might pass quite well for a case of organic disease. But notice that when she pushes the tongue out she throws the left face into a state of spasm, instead of the left side being weaker, as ought to be the case in organic disease. You will also notice that the contracture of the paralyzed upper limb is not that of organic disease. Look how flaccid the fingers are. Contrast this with the first case. Nor is there any absolute paralysis in this patient. This, then, is a case with glossolabiobrachial spasm. Such cases are extremely rare, and I am fortunate in being able to show you an example.

See, also, how her gait differs from that of organic disease. The first patient with organic hemiplegia dragged the sole of the foot along the ground in walking, as if she had an artificial limb. But this one dragged hers along in an absolutely effortless fashion. She dragged the dorsum. That is almost synonymous with saying that it is a functional and not an organic case. Finally, the state of the reflexes guides us in our diagnosis. The knee-jerks and supinator-jerks are equal on both sides, they are not exaggerated on the paralyzed side, and the plantar reflexes are both of normal type.

These are the chief points in this case which have led us to the diagnosis of functional paralysis, and I think that if you bear them in mind you will have no difficulty in recognizing most cases of functional hemiplegia.

The prognosis in these three cases is different. In the two organic cases the damage to the brain is irretrievable, part of its substance is permanently destroyed, and we cannot look for any more improvement than has already occurred. With regard to the functional case, matters are quite different. The prognosis is excellent as to recovery. Under appropriate treatment, isolation, massage, and special feeding this woman ought to get perfectly well. But with her previous history we should not be surprised if at some future date she were to develop a fourth attack of functional paralysis. The best way to prevent this will be to maintain her general nutrition in as high a state as possible.

SYPHILITIC HEMIPLEGIA; LEAD PALSY AND TRAUMA; ANOMALOUS GENERAL PARALYSIS; HYSTERICAL MUTISM.

CLINICAL LESSON AT THE PRESBYTERIAN HOSPITAL, NEW YORK CITY.

BY MAX SCHLAPP, M.D.,

Lecturer on Nervous Pathology at Cornell Medical School; Attendant Neurologist to the Dispensary of the Presbyterian Hospital, etc.

GENTLEMEN,—Our first patient, to-day, is an interesting example of the anomalous symptomatic picture that may occur as a consequence of cerebral syphilis. The patient came to the dispensary some weeks ago, suffering from weakness of the left arm and leg and left side of the face, and also with some speech disturbance. The weakness of the left side of the face is very noticeable whenever there is a change of expression in his countenance. The weakness of his leg can be seen in his gait, and the awkward method of carrying his arm shows that there is some loss of power there. This paralytic condition developed rather suddenly some ten months ago, and was at first much worse than at present. Improvement was satisfactorily progressive for some time after the first amelioration of symptoms began, but of late has almost entirely ceased. This makes the prognosis of the case rather doubtful.

The patient's history is of interest mainly from the stand-point of the etiology of the affection. He is a young man twenty-four years of age, well built, and with no signs of any tendency to deformity. He has for a number of years worked as a salesman in a wholesale house, and there is nothing in his occupation to suggest any predisposition to the occurrence of a nervous lesion. He confesses, however, to having contracted syphilis some three years before his stroke occurred. His syphilis was treated, and he seems to have had comparatively few secondary symptoms, and those not of a character to make him worry about his condition. It is not an unusual feature of the history of patients suffering from serious lesions of the nervous system as a consequence of syphilis, to find

that the original syphilitic process was not severe. This fact forms, indeed, the basis for the practical therapeutic rule that symptoms of severe degenerative lesions of the nervous system of obscure origin should always be treated by antisyphilitic remedies, even though there should be no history of syphilis, since so often in these cases the preceding syphilitic symptoms are of such a mild character that they may readily escape the notice of non-observant people.

About a year ago, the patient began to suffer from severe headaches, and then from increasing inability to use his limbs and especially his leg. For this he was under treatment for several weeks at Beth Israel Hospital in the lower part of the city. According to his wife, he was suffering at this time from brain-fever. This might arouse at least the suspicion of an infectious element in the case, with perhaps some symptoms of meningitis,—the usual pathological basis of the so-called brain-fever. As a rule, however, the laity class everything that affects the brain under the general heading of "brain-fever." In order to assure ourselves, the records of Beth Israel Hospital were consulted, and the clinical diagnosis was found to have been brain-syphilis. Very naturally, in answer to the inquiries of anxious friends at the hospital, this was not the diagnosis given; but it was stated that the patient was suffering from an affection of the brain. It will be found not infrequently that cases of brain-syphilis have had previous symptoms of the affection, though it may be hidden under some more euphonious name.

He seems to have recovered, more or less completely, from his symptoms, and to have begun once more to go about his work, when he was stricken one night with the paralytic condition, the remains of which we can see here at present. An examination of his condition shows that his eyes are unaffected by the pathological process, whatever it may be, and there is nothing in the history that would point to the existence of diplopia, or any other disturbance of vision at any time. His facial expression is quite markedly disturbed, and when we ask him to smile, his mouth is drawn considerably to the right side. When we ask him to put out his tongue, we find that it is protruded a little bit towards the left side, but the tendency towards either side is very slightly marked. His tongue is of firm consistency, not more flabby on one side than on

the other, is protruded without any trembling, and the patient seems to have good control over it.

An examination of the back of his mouth shows a seemingly healthy condition of the mucous membrane, but a distinct weakness of the muscles of the soft palate. When asked to say Ah or E, the uvula is but very slightly lifted; and it is evident that there are some disturbances of the muscles in this region. We have had his larynx examined by the dispensary laryngologist, but he found nothing of pathological significance in it. The patient is able to bring his vocal cords thoroughly into apposition, though their movements are somewhat slower than normal. This condition of his vocal cords is interesting, because the character of his speech disturbance might lead us to expect to find more interference with their action.

There is considerable disturbance of his power to talk, but this is not a true aphasia, nor has it any evidence of interference with the intellectual elements of the speech-faculty in it at all. The patient understands everything that is said to him. He knows what he wants to say in return, and is able to find the words, but cannot utter them properly. The speech disturbance is what is known as dysarthria, that is, failure to articulate properly. The patient's condition in this respect has improved very much since the stroke, and he seems to be gaining control over the complicated nervous mechanism that rules over the coördination of muscles required for proper articulation.

His intellectual faculties are practically undisturbed. He is able to tell the day of the week and month, and to give various dates for events in his own life, so that even his memory, the lowest of the intellectual faculties, is not impaired. He multiplies reasonably well, knows where he is, what the hospital address is, what his home address is, and seems practically in every respect normal. It is true he has a tendency to a neurotic condition, to times of discouragement and alternate excitement, but these are hysterical symptoms superadded to his fundamental nervous condition rather than the direct result of the organic nervous lesion from which he is suffering.

After all, it must not be forgotten that the patients who have a real organic nervous lesion often have an accompanying neurotic condition of all the nervous system that provides a good basis for hys-

terical manifestations. If we consider that people in good health may have hysterical symptoms, there is so much the more reason for suspecting the presence of hysteria in true organic nervous affections. This is often forgotten by students of nervous diseases when they are beginning their work in this specialty, and so, passing neurotic symptoms of a functional character are presumed to have a significance that does not really belong to them, and the consequence is that the problem of diagnosis becomes apparently more complex than it is in reality.

The treatment of a case like this consists, of course, in the administration of antisyphilitic remedies. When the patient first came, these were at once prescribed. The mercury was administered, as is usual in the dispensary service here, by inunctions. The patient did not bear the drug very well, however, but developed serious gastro-intestinal troubles, while his skin did not react kindly to the mercurial applications. After several weeks, then, the use of mercury was given up, and he was placed upon the iodides alone. At the present time he is taking sixty grains of potassium iodide three times a day. His condition has markedly improved, though of late the improvement has not been very steady. Such interruptions in the convalescence, however, are not unusual during a course of antisyphilitic treatment. They should not be allowed to prove too discouraging, nor force the physician to the idea that these remedies will not do any more good. After a certain period of apparent obstinacy to treatment, amelioration of symptoms will frequently be noted once more, and may progress to a very encouraging degree.

The ultimate prognosis in this case, as regards complete recovery, is not, however, very favorable. The speech disturbance has continued so long that it seems not improbable that some difficulty of articulation will remain all during the rest of the patient's life. It is important that the treatment of syphilis of the nervous system should begin as soon as possible after the first symptom has manifested itself, and should be pushed vigorously until the full effect of the remedies is obtained. The delay of a very few days may mean the establishment of a degenerative process in important nerve-structures in which repair is hopeless.

It is very important, then, that those who treat patients for primary and secondary syphilis should impress upon them the

dangers of the subsequent development of nervous syphilis, and tell them of the importance of noticing such preliminary symptoms as headache, which is usually worse at night, or a tendency to double vision, or awkwardness in the use of the limbs that may occur. If the patient realizes what the significance of such symptoms may be, he will not delay consultation with a physician when the first signal of the approach of nervous involvement becomes manifest. Besides, he will not be apt to leave his physician in doubt as to the condition that really exists by failing to tell him, or perhaps by even denying, the presence of syphilis in his past history.

In order to keep syphilitic patients from worrying as to the possible consequences of their malady, physicians sometimes think it advisable to assure them, after their secondary symptoms have passed over, that now they are all right. Such assurance is liable to do much more harm than good, and every patient has the right to know, and every physician is under the obligation to tell candidly, the danger that exists of the subsequent development of nervous or visceral tertiary lesions. At least one-third of all syphilitics have serious late symptoms and complications of one kind or another. It may be objected that this will prove a needless source of worry to the majority of syphilitic patients, but it is better that all should suffer this discomfort than that not a few should develop irreparable nervous lesions, or even fatal complications, because they have not been warned of the possibilities in store for them, and have failed to have recourse to treatment in time.

Our next case, to-day, is a man some forty-two years of age, who has been working at the occupation of painter very faithfully for about twenty years. He has never had any symptom of lead poisoning, and insists that he has enjoyed excellent health all his life. Careful inquiry shows that he has not suffered from constipation, nor has he had any pains in the abdomen that might lead us to think that he had suffered from even a mild form of lead colic. His mouth is reasonably clean for men of his class, and there is no appearance of the bluish lead-line on the gums. He would seem, then, to have escaped entirely from the effects of constant contact with lead. It would appear, indeed, that he was one of those fortunate individuals, occasionally met with, who are immune to the manifestations of lead poisoning.

One of the most interesting features of lead poisoning is the rôle that idiosyncrasy plays in the precocious or delayed development of the symptoms of plumbic toxæmia. There are some persons in whom the slightest contact with lead, if continued for even a short period of time, is sure to be followed by distinct symptoms of lead poisoning. In others, after a short interval, though there have been very few preliminary symptoms, there is a sudden development of acute stigmata, that shows that lead has been accumulating in the system for some time, and is capable of producing serious effects. Lead colics particularly are sometimes almost fulminant in character, and come on with scarcely any warning, making a difficult diagnostic problem for the practitioner, who has no history of previous symptoms in the case to guide him, and may consider that the intensity of the abdominal symptoms points to some serious intra-abdominal lesion, requiring, perhaps, surgical intervention.

When we inquire what our patient comes to be treated for, he holds out for our inspection a typical drop-wrist in his right hand, that would at once lead us to ask him his occupation, if we did not already know it. It is evident that he is suffering from the paralysis of the extensor muscles of the hand which occurs so frequently during lead poisoning, and is so characteristic of it. This paralysis, in his case, is more severe than usual, and even the supinator longus, which is often spared in lead palsies of the forearm, is involved in this patient. The paralysis of the supinator longus can be tested in such cases by asking the patient to supinate his hand, that is, to turn it over so that the back of the hand rests upon the table or on the patient's knee when sitting down.

Our patient in this case is able to swing his hand around in a certain way, so that his knuckles rest upon the table, but it is very evident that this movement is not accomplished by the ordinary mechanism of supination. He is able, by swinging the forearm by means of the muscles of the upper arm, to turn the hand around, so that an imitation of the movement of supination is accomplished. This movement is interesting, because it shows that in a careless examination it might seem as though supination were present, when it is really absent. In this case, it is evident that the supinator longus is also involved.

The history of the development of the condition is the most striking feature of the case. While on the street, some days ago,

the man slipped and fell, and tried to save himself by means of his hand. He sprained his wrist rather severely, but was able to use his fingers rather well, and suffered only from some tenderness on motion of the wrist-joint. Gradually, however, he noticed loss of power in his hand and failure to extend his fingers. He woke up one morning to find the condition of drop-wrist, as it now exists, fully developed. The condition has persisted ever since.

The history of this patient is quite typical of many of the nervous conditions that develop as the result of chronic toxæmias of various kinds. It is not unusual to find that there has been no symptom of the toxæmia manifest until trauma, or some emotional strain, or some sudden nervous shock such as fright, causes the nervous system to give way at some part. Despite the presence of irritating substances in the circulation, or in important tissues, the nerves have been doing their work with perfect regularity. As the result of the shock to the system, however, they fail to be able to accomplish their function in the presence of serious disadvantages, and so the symptoms of the toxæmic nervous condition become manifest.

This is true not only for lead poisoning, but also, and perhaps even more strikingly, for chronic alcoholism. It is not an unusual thing to find that a man who has for years indulged to excess in alcohol, without suffering from severe nervous symptoms, develops delirium tremens shortly after some serious injury, that compels him to keep his bed for some time. This is so well recognized by physicians in attendance at large hospitals, that when patients are brought in suffering from fractures of the leg, for instance, they make it a point to inquire as to the previous habits of the individual with regard to alcohol, in order to be able to take the necessary precautions so as to prevent the patient from injuring himself in case delirium tremens should develop. In these cases, it is not unusual for the symptoms to come on almost without warning, or the true delirium tremens is preceded by only a few nervous symptoms that may not arouse suspicion of the serious condition that is to follow.

Alcoholic patients who suffer from neuritis of the leg with toe-drop not infrequently have their serious symptoms develop after a sprain of the ankle or some injury to the foot. These cases are very similar in their origin to this case of drop-wrist from lead

poisoning, because of the fact that they frequently give no preliminary symptoms, and no warning of the irritative nerve conditions that exist until after the trauma leads to the development of the characteristic paralysis of the muscles.

It is well, then, in cases where there is any history of alcoholism, or where patients are in such an occupation as that of painter, plumber, or tinner, or any other in which lead is handled freely, that if anomalous symptoms develop after the occurrence of trauma, sprain of the wrist, or of the ankle, or the like, or even a fracture, the possibility of the existence of an associated neuritic condition should not be forgotten. As a matter of fact, in slight amount, these neuritic manifestations are much more frequent than used to be thought, and their recognition is important for the proper treatment of the case.

The treatment of a case like this consists mainly in the encouragement of the elimination of the lead in the tissues. The drug that accomplishes this purpose best is the iodide in some form, and potassium iodide is the one most used. This forms a compound with the lead in the tissues which is soluble, and so the lead comes to be gradually eliminated. Most of the elimination is done through the kidneys, and therefore it is important at this time that the man should drink freely of water, in order to keep his urine dilute and free from irritating properties for other reasons. It is also important that the use of alcoholic liquors and other substances that may prove irritating to the kidneys at this time be avoided, because it must not be forgotten that the elimination of lead leads to certain sclerotic changes in the kidney which resemble those that occur in gout. As far as possible then, the kidneys should be saved irritation during the period of lead elimination, and for this a bland, unirritating diet, consisting not too exclusively of meat, but containing an abundance of cereals and of milk, should be recommended.

With regard to the local conditions in the muscles, electricity will do something to restore them to their tone as soon as the lead has been somewhat eliminated from the nerves supplying them. For this the slowly interrupted current should be employed, at a strength just sufficient to cause contraction of the muscles, but without occasioning the patient any discomfort. The continuous current may also be used for a short period each day, but its general

tonic effect upon the tissues, owing to the lack of exercise of the parts while the paretic condition exists, causes their general vitality to become somewhat lowered.

The most important recommendation to patients is with regard to the practice of the proper prophylactic precautions, so as to avoid lead absorption. It is generally recognized now that the plumbic toxæmia occurs, not so much because of the absorption of lead through the skin, as through the digestive tract. Workmen who handle lead in various forms neglect to clean their hands thoroughly before taking food. The result is that small but appreciable amounts of lead are taken at every meal. Sooner or later, this leads to the deposit of lead in the tissues and the occurrence of the symptoms of lead poisoning. It is not sufficient for workmen merely to casually wash their hands in soap and water, but they must thoroughly scrub them before eating and especially must clean out beneath the nails. It is important, even, to suggest that the nails should be intentionally worn rather short, so as to prevent the accumulation, as much as possible, of the lead beneath them. It has been shown by the application of sensitive chemicals to the hands that, even after a week or ten days, painters and others who handle lead may still have traces of the metal on their hands.

It is important that the bowels of those who work in lead should be kept freely open. For this purpose the best remedial measure is the use of magnesium sulphate, ordinary Epsom salts. The sulphate forms a compound with the lead present in the intestines, and this is carried out much more simply than if it were left to the chances of absorption. Enough of the drug should be given to cause a soft stool every day, but not more than this, or the patient may be weakened. Patients who are suffering from nervous lesions due to lead poisoning, and to whom potassium iodide is being given, should also be advised to take magnesium sulphate. Directions have been given to our patient to take a teaspoonful of Epsom salts every morning, or less according to its action on him.

There are some people who are very insusceptible to lead, and who, notwithstanding the occurrence of one such nervous accident as we see in this case, are still able to continue their occupation in contact with lead, if the precautions we have suggested are taken. There are others, however, who are so susceptible to plumbism, that one accident succeeds another, and for these it is

important to be given the advice to change their occupation as soon as possible. In the present case, our patient seems quite insusceptible, and so we shall await further developments before suggesting change of occupation, unless he finds it reasonably easy to do so without serious detriment.

Our next patient presents an extremely interesting set of symptoms, all the more worthy of our study because it is not very clear just what may be the primary factor in their production. He is, as you see, a hunchback, and suffered from Pott's disease of the mid-dorsal vertebræ when he was less than five years old. Since then there has been no recurrence of any symptoms of the tuberculous process in his spinal cord, nor has there been any sign of tuberculosis in any other part of the body. He is now in his sixtieth year, so that we may practically dismiss all thought of the old Pott's disease as the causal factor in the case. Besides, there is no special tenderness over his spine at the point of deformity, and no sensitiveness to jarring or rough riding on cars, or the like, that would serve to indicate a tendency to recrudescence of the tuberculous affection.

His symptoms are briefly these: he suffers from a series of attacks, which recur a number of times a day, and are marked by a temporary lapse of consciousness, after which he feels somewhat depressed. These are not unlike epileptic attacks, inasmuch as they are accompanied by a sensation that at least simulates an aura. A feeling of oppression begins in the lower part of his abdomen, and seems to him to mount upward until he is overcome. Sometimes the peculiar feeling in the abdominal region seems to spread to the lower limbs, and makes them feel more or less powerless before the lapse of consciousness takes place. He insists, himself, that he loses consciousness entirely, though it is only for a few moments.

While this would seem to indicate that he is suffering from epileptiform attacks, there are certain well-known stigmata of epilepsy that are entirely lacking. A glance at his tongue shows that there are no scars upon it, and he himself says that he has never bitten his tongue during these attacks. There is no history of his ever having fallen before he was in such a position that his fall would not be serious. He has never been suddenly taken by an attack when going down or coming up stairs, and in general it is

clear that he does not lose consciousness so suddenly as not to be able to make due provision for his safety. He utters no cry when his attack comes on, and the loss of consciousness is at most only momentary.

These attacks have been noticed only during this last year. There is no history of alcoholism nor of syphilis in the case, and idiopathic epilepsy developing as late in life as this is so rare as to be practically out of the question. There are certain symptoms that show, however, that there is some serious affection of the central nervous system at work. Besides his attacks, his wife says that he has changed greatly in disposition during the last few months, and that from being very faithful in taking care of his work as a shoemaker, he has practically been compelled to give it up entirely. He complains himself of loss of memory, and his wife says that he frequently puts things away around the house, and is utterly unable to remember where he has placed them after only a few hours' interval.

A more serious set of symptoms is disclosed by the fact that his wife complains that he has become changed from a very thrifty man into one of somewhat prodigal disposition. He not infrequently buys things for which he has no use, and has made expensive purchases of more or less useless articles for her and for the house. His friends also have noticed this change in his disposition, and have remarked that he was much freer with his money than he was a few years ago. This change of disposition would raise a suspicion as to the possibility of incipient general paralysis. If we ask him the ordinary questions, however, we find that his memory for dates and places is reasonably good, that he is able to multiply well, and that, while there is a tremor of the lips and tongue, there is no difficulty in the articulation even of difficult phrases.

The one objective symptom in the case that is very significant is the presence of Argyll-Robertson pupils. There is complete failure of the reaction to light. This is a symptom that might easily be missed, but it seems to furnish a key to the diagnostic problem. In connection with the other symptoms, the recurrent attacks, the tremulous lips and tongue, the changed disposition with marked tendencies to prodigality and forgetfulness, this involvement of the pupil seems to justify the diagnosis of progressive general paralysis. It is not a typical example of the condition, but rather one of the

anomalous forms that sometimes run a long course, and the remissions of which give opportunities for the announcement of so-called cures.

There is no history of syphilis in the case, but there is indication for the employment of antisyphilitic treatment in all such cases. For many months our patient was on the iodides, but failed to manifest the slightest improvement, though there was good tolerance for reasonably large doses. The only remedy that has proved helpful in limiting the number and severity of his epileptiform attacks is the bromides.

These patients must be kept in as good general condition as possible. Their appetite must be encouraged, their despondency must not be allowed to interfere with the taking of food regularly, and their bowels must be carefully regulated. The use of nutrient remedies is often indicated, and our patient always feels better while he is taking malt and cod-liver oil. It is probable that the tendency to progress of the nervous degeneration may be delayed by proper care of systemic metabolism.

Our next patient is a young woman who is unable to speak above a whisper. When she first came to the clinic some months ago, she was absolutely unable to utter a sound of any kind. She evidently understood everything that was said to her, and made efforts to talk, but was utterly unable to articulate even a syllable. Besides this aphasia there was a condition of double ptosis. She could not see anything except by placing her head very far back, when she was able to perceive obscurely beneath the lowered curtain of the eyelid. The history of the case very soon showed what the probable condition was. The girl is known to be very nervous. There was, besides, a distinct neurotic history. A short time before her condition of mutism developed, she had been very much disturbed by the death of a brother to whom she was very much attached. A few days after the funeral she went to bed in normal health, and woke up with her condition of mutism fully developed and with the ptosis that I have described. The condition was evidently one of hysterical paralysis of the orbicularis muscles. Other means of treatment having failed, she was hypnotized, and, under the influence of suggestion during the hypnotic state, gradually recovered the use of her levator muscles. Her voice, however, did not return, in spite of suggestions directed to overcome her mutism.

She accepted very well all suggestions made in the hypnotic condition, but could not be brought to the resumption of the proper use of her vocal cords. She was engaged to be married, and as her fiancé was anxious that the marriage should take place, it was suggested that the change in her condition incident to matrimony might prove of therapeutic benefit. This surmise proved correct. The day after her marriage, her voice was restored sufficiently to enable her to talk and whisper as you hear her now. Since then, however, very little improvement has taken place. She has learned to whisper without difficulty, but she has not been able to vocalize any of her words.

We have tried the use of hypnotism, with the idea of bringing about improvement in her voice, but so far without success. It was rather difficult at first to bring her under the influence of hypnotism, but she goes into the hypnotic condition without difficulty now. At my suggestion that the waves are approaching, you see she puts on a frightened look, and cowers away from the direction in which I point them out. When I insist that they are coming nearer, and that she must save herself, she proceeds to get up on the chair. When I tell her that she will have to swim, she moves her arms as if to strike out in swimming, though she remains standing. When I tell her that she must call for assistance or she will surely be drowned, an intensely scared look comes into her face, and she evidently makes a supreme effort to call out. She is able to emit, however, only a hoarse whisper for help, and despite the fact that she is thoroughly under the influence of hypnotic suggestion, and is completely oblivious of her surroundings, so that she is firmly persuaded that she is in the midst of great danger, the neurotic paralysis of her vocal cords does not let up, and much as she would wish to, she is unable to utter a loud cry.

These cases of hysterical mutism are not very rare, and the diagnosis of them is not difficult, as a rule. They usually occur in persons of distinctly neurotic disposition with a history of previous manifestations of more or less serious nervous symptoms, and usually a marked neurotic heredity. The mutism develops suddenly, and usually without any lesion of the throat or larynx. In persons that have been affected with hysterical mutism, it may happen that a severe sore throat, an acute tonsillitis, or the like, may

bring about a recurrence of the inability to articulate. These cases are often very puzzling to the general practitioner.

An examination of the larynx in cases of hysterical mutism usually shows no local pathological condition, and the only thing noted is a paralysis of one or both vocal cords on the abducted position. The only hope of successful treatment in the case is by producing sufficient mental influence upon the patient, so as to enable her to regain control of the laryngeal muscles. Sometimes this can be accomplished almost at once by bringing the patient into a hypnotic condition and giving her the direct suggestion that she shall talk, or still more effectively suggesting to her that she is in danger, and insisting that she must call out for help. At times patients will be able to call out loudly while in the hypnotic trance, and yet either be mute or unable to speak above a whisper after they have come out from under the influence of the hypnosis. Sometimes, as in the present case, hypnosis will fail entirely to affect the condition.

As a rule, patients are influenced for the better only by some very strong mental influence. In this case, her marriage proved effective to a certain degree. Not infrequently, however, the voice is only gradually recovered. In married women sometimes the emotions incident to labor, and especially the birth of a child, may prove effective. Patients who have once suffered from mutism are likely to have recurrences of the affection. These may supervene suddenly as in the original attack, and are usually the result of some emotional strain. These recurrences are easy of diagnosis, but are apt to prove quite as obstinate to treatment as the original condition. Such patients should be warned of the necessity for avoiding stresses of emotion and all excesses, and of the need of a quiet, restful life.

Surgery

ANATOMY OF THE INGUINAL REGION AND THE RADICAL CURE OF INGUINAL HERNIA.

SPECIAL ARTICLE WITH ILLUSTRATIONS FROM DISSECTIONS MADE AT THE JOHNS
HOPKINS MEDICAL SCHOOL.

BY M. F. FALLON, M.D.,

Attending Surgeon to St. Vincent's Hospital, Worcester, Mass.

OF late years it has come to be very generally realized that operations for the radical cure of hernia constitute a very encouraging feature of thoroughly conservative surgery, and it is evident that these operations, far from being any mere fad of the moment, will surely occupy even more attention in the near future than they have up to the present time. The statistics of operations for the radical cure of hernia show that the procedure is attended, in careful hands, by but very slight mortality; and the faithful following up of cases after operation shows that the number of relapses when properly chosen methods of operation are applied, represent only a very small proportion of those operated upon.

Success in the performance of an operation for the radical cure of hernia depends mainly upon an intimate knowledge of the minute details of the anatomy of the structures of the abdominal wall and of the abdominal viscera immediately in relation to them. Of course, it is understood that surgical technic and a thorough mastery of the principles of asepsis are necessary preliminary qualifications for the performance of an operation that involves such important structures.

I have considered, therefore, that a presentation of dissections of the inguinal regions, made with special reference to the demonstration of the various tissues that are encountered in an operation for the radical cure of hernia, would have a special interest and a very practical importance for practising physicians generally.

Inguinal hernia is chosen because it occurs more frequently, and because, if a radical operation is done for the condition before any complications have developed, the accomplishment of a radical cure is not only easy, but there is almost perfect assurance that recurrence will not take place. The dissections are made with special reference to Bassini's method, because it is now generally acknowledged that the operation invented by the distinguished Italian surgeon is founded upon sound anatomical principles, gives as good if not better results than any other method of radical cure so far suggested, and is employed at the present time by far the vast majority of surgeons all over the world.

The operative technic for the Bassini operation is too well known to require detailed description here, but I recall the main features of it in order to make the successive steps in the dissection more clear. The skin incision extends from about the level of or a very short distance below the anterior superior spine of the ilium to the external inguinal ring. The retraction of the edges of this incision at once exposes the aponeurosis of the external oblique muscle. If a wider exposure than can be obtained by this incision in the ordinary relaxed condition of the cutaneous tissue is desired, it may be obtained by retraction downward of the lower angle of the incision. It is not advisable, however, to extend the incision itself any further downward. The inguinal fold is an extremely difficult place in which to maintain perfect asepsis, and the proximity of the lower end of the incision to the urinary organs makes clear the necessity for very special precautions in this matter. During the course of the incision of the cutaneous and subcutaneous tissues, the superficial epigastric vessels and one or more branches of the superficial external pudic artery, lying on the deep layer of the superficial fascia, are severed. The lower branch of the latter sometimes lies below the inferior limit of the incision, and escapes being cut. These vessels are the largest found in the operation and they should be at once tied. The external oblique muscle is recognized by its coarse, tendinous fibres. The external abdominal ring is freed; the handle of the knife, or a director, is inserted in the ring, under the aponeurosis of the external oblique. The external oblique is now cut on the director, and both edges well freed from underlying tissue. The cord-mass, including cord and sac, is freed from its bed by blunt dissection,—guarding against

injury to the vas deferens and to the ilio-inguinal nerve. The sac is separated from the cord, beginning preferably at the internal ring. The separation must be continued even to the parietal peritoneum immediately surrounding the internal ring. The sac can usually be isolated with the handle of the knife and with gauze.

If there are dense adhesions, and it is necessary to use the sharp parts of the scalpel, the tissues should first be made tense and carefully inspected. Important structures may easily have become dislocated in the neighborhood of a long-standing hernia and be cut inadvertently. Sometimes in an old and previously inflamed hernia the fundus of the sac may be so intimately adherent to the surrounding hypertrophied tissues that it can be separated, if at all, only with the greatest difficulty. In such cases the distal part of the sac may be left *in situ*, undisturbed. Careful conservatism in this matter will not, as a rule, endanger the success of the operation, and it will save laceration of tissues and needless risk.

When the sac is freed, the fundus is widely opened and the contents inspected. After being freed from its contents, the empty sac is as gently as possible pulled out of the internal ring and ligated at its abdominal end with medium-sized plain catgut. The ligation should be so high that no peritoneal depression leading towards the former hernial track is left. In ligating, the operator may insert the index-finger of his left hand through the neck of the sac into the abdomen. The assistant then ties the sac around the finger and brings the knot firmly home, as the operator withdraws his finger. This precaution will effectually prevent any portion of the abdominal contents from being included in the ligature.

The ligature is rendered absolutely safe by transfixing the sac and carrying the transfixed ligature once or twice around its pedicle. The sac is now amputated at a safe distance below the ligature, and the stump should be carefully inspected before being allowed to slip back finally into the abdomen. The isolated cord is lifted up, and held towards the abdomen by means of a tape. The canal is freed from loose fat and excess of tissue. The next procedure, the most essential of all, is to construct a new, firm posterior wall, by sewing the edge of the united internal oblique and transversalis muscles and the underlying fascia to the lower free edge,—the so-called shelf of Poupart's ligament. Every step should now be taken under the careful guidance of direct vision. Nothing should be done in

the dark, for fear of embarrassing and possibly fatal accidents,—from injury to surrounding important structures, to the deep epigastric or femoral vessels, or to the abdominal contents. A wide exposure of the whole canal is made by freeing the cut edges of the external oblique by blunt dissection, on the one side as far as the rectus, and on the other to, and including, the shelf of Poupart's ligament.

The edge of the united internal oblique and transversalis muscles, with the underlying fascia, can be everted with forceps; or the finger may be inserted under the muscles as a guide to the needle. The shelf can also be lifted with forceps, and so sutured. There should be no tension, or the main purpose of securing firm immediate union of the tissues will almost surely be prevented. The periosteum of the os pubis should, as a rule, be included in the suture. The suture should, however, be so inserted as not to include the hypogastric nerve. Coley has suggested the use of an additional suture not so deep as the others, which should be inserted above the internal ring through the internal oblique muscle and the upper exposed part of Poupart's ligament, though not including the so-called "shelf" of the ligament. An anatomical reason for this is the partial separation of the fibres of the internal oblique above the internal ring, while below there is a complete separation because of the descent of the testicle.

The cord is now placed on the newly constructed floor. The field of operation is thoroughly dried. The cut edges of the external oblique are reunited. Just enough room is left at the site of the former external ring for the free passage of the cord. The firm deep layer of the superficial fascia is separately sutured, and finally cut edges of the skin are brought into close apposition; sterile gauze is placed on the wound, then a spica bandage. A plastic bandage extending from the costal margin to the middle of the thigh favors immediate primary union by completely immobilizing the parts. This same precaution secures postoperative asepsis.

The patient should stay in bed at least two weeks, and the wound should be inspected on the eighth or ninth day. The care of the patient, before and after the operation, should be the same as in all laparotomies. If the operation is successful, the patient should not wear a truss afterwards.

The accidents most frequently occurring in the field of operation are: injury to the femoral vein or to the deep epigastric vessels; to the vas deferens or its blood-vessels, causing atrophy or gangrene of the testicle, with loss of function; to the ilio-inguinal or iliohypogastric nerves, causing subsequent paralysis; and, finally, to the abdominal organs in the sac. Almost every abdominal organ has been found in an inguinal hernia, though those most frequently seen are the small intestines and the omentum. The part of the bladder covered by the peritoneum, as well as the portion not covered by it, may be present (similarly, the ascending and descending colon, following their normal anatomical relations). The bladder-wall may be thin, simulating the sac; or surrounded by fat, so that in releasing adhesions it may easily be torn. This is especially to be borne in mind in direct hernia. When the appendix is in the sac, it is, for aseptic reasons, best disposed of by inversion rather than by amputation.

Suture-Material.—The contentions of to-day over suture-material in hernia operations remind one of the bitter disputes on the same subject even in the time of Pierre Franco, a distinguished French surgeon of the latter part of the sixteenth century. He branded “plusieurs maistres de notre art” (many masters of our art of surgery) as “brigands” or “affronteurs,” who, in hernia operations, used hemp, linen, or silk threads, instead of gold wire, favored by him. Both absorbable and non-absorbable suture-materials have their advocates; the absorbable sutures most used being kangaroo-tendon and chromicized catgut; while the favorite non-absorbable materials are silver wire and silk. Non-absorbable sutures are admittedly foreign bodies. They are apt sooner or later to cause suppurating sinuses, and, in doing this, invalidate the plastic union of the tissues on which depend the future strength of the abdominal wall at this point. They thus favor a relapse. If absorbable tissues can give a firm union before they are absorbed, they should be the suture of choice.

The chief objection to animal sutures is due to failure to properly sterilize them. They can be perfectly sterilized. The writer prepares catgut by the formalin method, and stores it dry in glass tubes, in which the air is rarefied by heat at the time of sealing. Moisture, essential to bacterial life, is absent; and this method is probably preferable to storing in alcohol, or any other liquid. Prop-

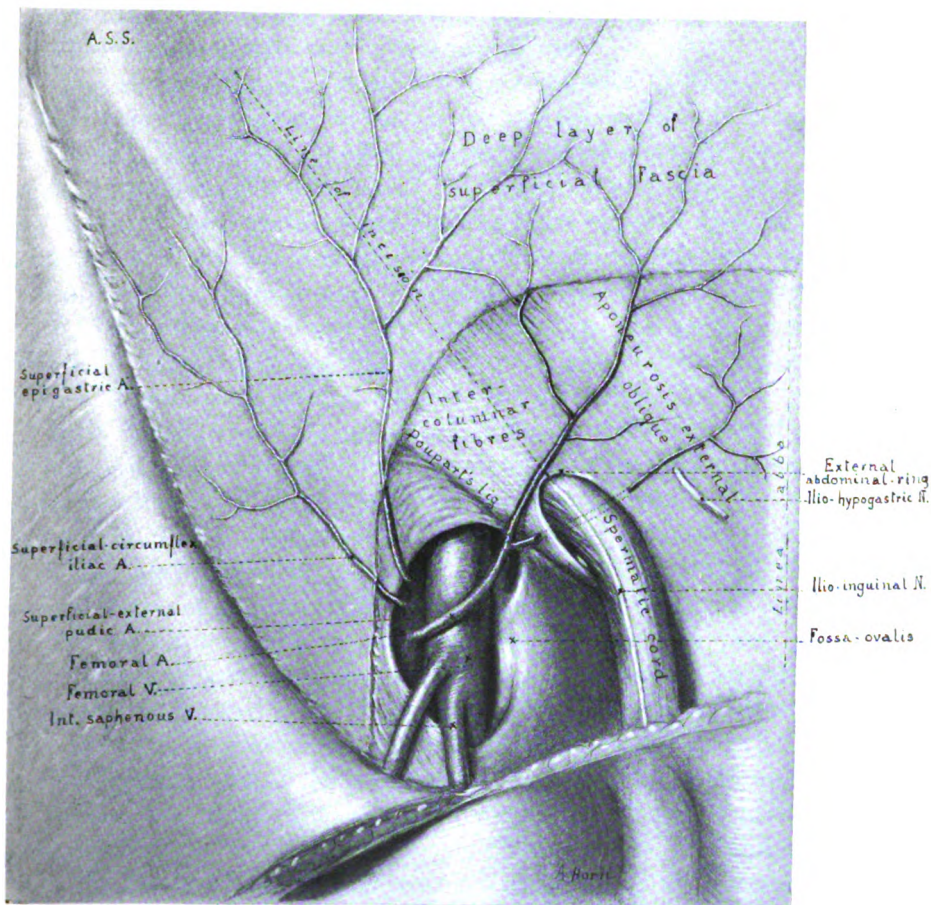


FIG. 1.—Relation of blood-vessels and nerves to line of incision in the radical cure of inguinal hernia.

erly chromicized catgut of medium size (not larger than No. 1) will give a firm union in the deep layer of the hernia operation, and in due time be absorbed. An interrupted suture is generally used for this layer. A medium-sized plain catgut is suitable for the edges of the external oblique; a fine plain catgut for the firm layer of superficial fascia, and also for the skin. All these layers can be rapidly sutured with the button-hole suture.

Care should always be exercised in the selection of cases. The radical cure should not be advised if for any reason there is danger to life, or if there are not good prospects of a permanent cure,—*e.g.*, in flabby abdominal walls, where frequently several large herniæ are present. Many skilled surgeons, in a large series of cases, have a mortality of less than one per cent., and almost no relapses. The mortality is generally due to pneumonia, common to all laparotomies. Unfavorable subjects for operation are old people with bronchial catarrh, patients with emphysema, heart-disease, kidney-disease, diabetes, and general debility, and alcoholics.

Most surgeons exclude cases for radical cure when under four or over sixty years of age. If more attention were paid to hernia in infants there would be fewer hernia operations in adults. Kocher has shown that a surprisingly large number of infants are born with an open processus vaginalis. With proper treatment this nearly always closes during the first few months of life.

No major operation is safer and more satisfactory than the radical cure of hernia rightly done; but no other single operation has been so productive of fatal results to patients, and loss of reputation to surgeons when unskilfully or carelessly done. The most important part of the preparation for the skilful performance of radical hernia operations, after a proper knowledge of general surgical technic, is, as has been said, a thorough acquaintance with the layers of tissues that are met with during the operation, and of which use has to be made in forming the new inguinal canal. With the idea of facilitating this knowledge for those who are not in favorable circumstances for dissection, the following illustrations have been prepared with the greatest care as to exactness and completeness of detail.

The illustrations by Mr. Horn made from dissections by the writer at Johns Hopkins show very beautifully the anatomical relations of the structures. Fig. 1 gives the relations of the blood-vessels and nerves to the skin-incision.

the structures. Fig. 1 gives the relations of the blood-vessels and nerves to the skin-incision.

The skin and superficial layer of superficial fascia are reflected downward, exposing the firm deep layer of the superficial fascia. The blood-vessels are on this layer.

A window in the deep layer of superficial fascia exposes the external abdominal ring and the spermatic cord; the ilio-inguinal nerve, accompanying the spermatic cord and emerging from the external abdominal ring, to be distributed to the integument of the inner part of the thigh and to the scrotum; the hypogastric branch of the iliohypogastric nerve, perforating the aponeurosis of the external oblique, to be distributed to the integument of the hypogastric region.

The second illustration presents the next step in the operation, and the structures it brings into view. The aponeurosis of the external oblique is incised and the cut portions reflected, exposing the internal oblique and the cremasteric muscles and the inguinal canal. The upper wall of the inguinal canal formed by the united border of the internal oblique and transversalis muscles can be well seen. The hypogastric branch of the iliohypogastric nerve is seen on the internal oblique muscles.

The third illustration shows the rear portion of the inguinal canal exposed by the removal of a portion of the cord. In order to secure this view the transversalis fascia is removed and the lower portion of the external oblique aponeurosis reflected downward. The lower fibres of the internal oblique and transversalis muscles are detached from Poupart's ligament and reflected medianward, over the edge of the rectus muscles. The spermatic cord can be seen emerging from the internal abdominal ring and passing over the deep epigastric artery. The canal portion of the spermatic cord has been excised. Poupart's ligament, Gimbernat's ligament, and Colles's ligament are all shown as parts of the aponeurosis of the external oblique. The outer margin of Gimbernat's ligament forms the inner boundary of the crural ring.

The lowermost fibres of the transversalis muscle spread, and have a fan-like or triangular attachment to the upper surface of the os pubis and to Poupart's ligament. On the posterior aspect of the muscle and towards the median line are tendinous fibres arising from the edge of the rectus (*Falx inguinalis*). On the

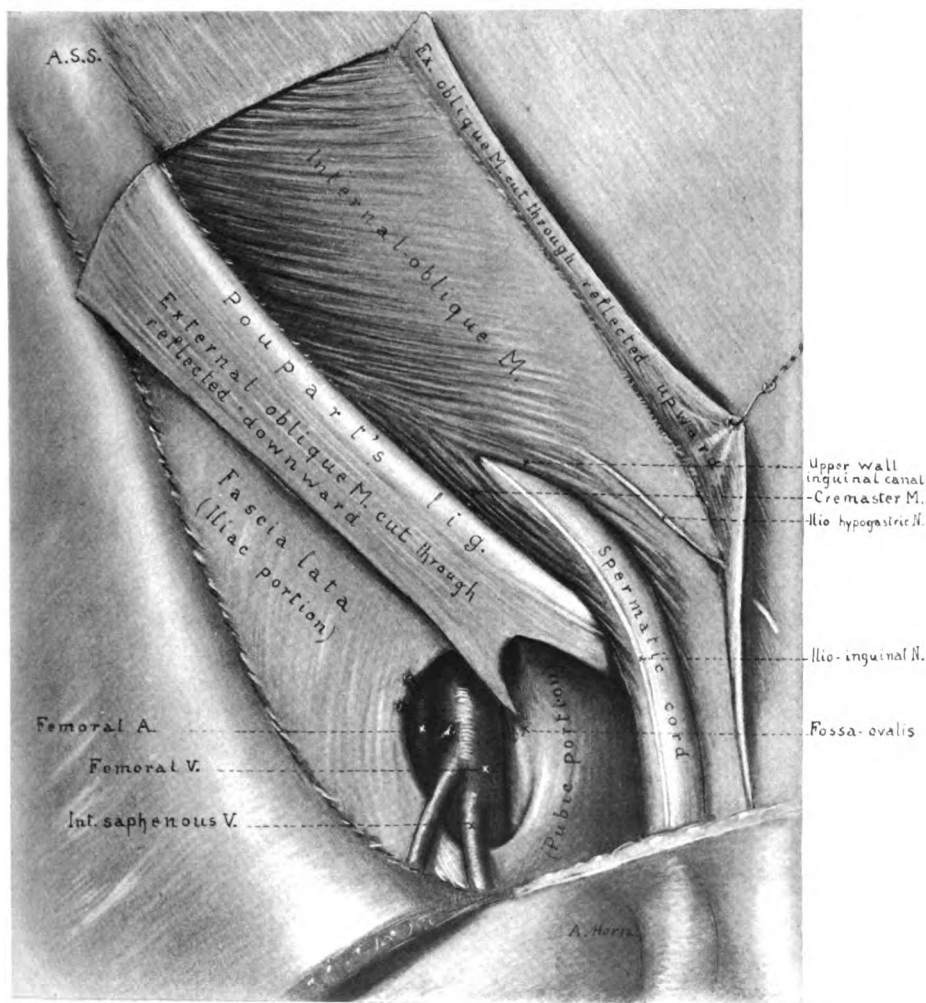


FIG. 2.—Right inguinal canal under normal conditions, showing muscular and fascial relations.

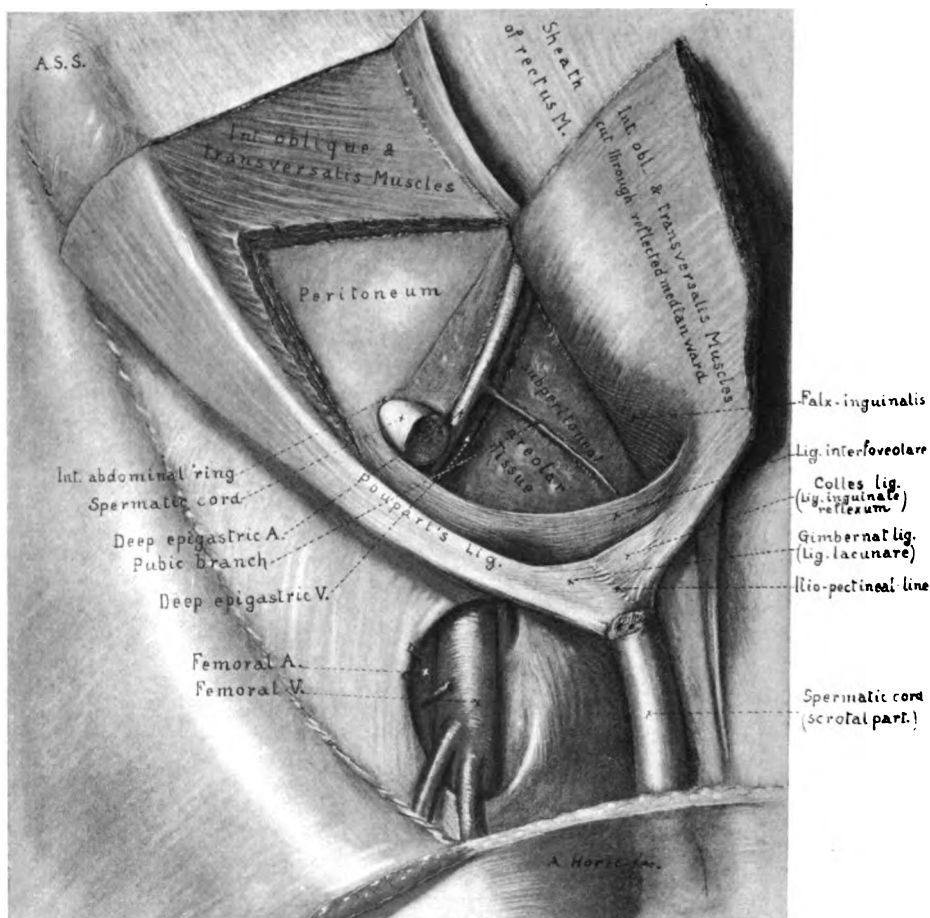


FIG. 3.—Floor and neighboring deep structures of inguinal canal, showing tissues that come into view in the operation for the radical cure of inguinal hernia.

anterior aspect of the muscle are fibres arising from the internal oblique. Hence this triangular plate is musculo-tendinous. It lies posterior to the inner pillar of the external abdominal ring, and, with the ligamentum interfoveolare, enters into the formation of the posterior wall of the inguinal canal.

The ligamentum interfoveolare is a thin, curved band of firm semi-tendinous fibres, arising from the under surface of the transversalis muscle, near its insertion, extends as high up as the internal ring, and is attached to Poupart's ligament.

Poupart's ligament gives part attachment to the fascia lata. This attachment and the manner of insertion of the fibres of Gimbernat's ligament into the pectineal line cause a rolling inward or reflection of the lowermost fibres of Poupart's ligament, surgically known as the "shelf." This shelf, the whole length of the inguinal canal, is free from attachments on its upper and under surface, and forms almost a right angle with Poupart's ligament. The spermatic cord rests on this shelf in the lowermost portion of the canal, and here the shelf is about one-half an inch wide. In Fig. 3 the shelf corresponds to the part marked "Poupart's lig."

The deep epigastric artery arises from the external iliac, passes behind Poupart's ligament and the ligamentum interfoveolare, ascends obliquely upward and inward along the lower and inner margin of the internal abdominal ring, beneath the spermatic cord.

THE SURGICAL TREATMENT OF HÆMATEMESIS FROM GASTRIC ULCER.

BY BERKELEY G. A. MOYNIHAN, M.S. (Lond.), F.R.C.S. (Eng.),
Assistant Surgeon, Leeds General Infirmary.

DURING the last two or three years there has been considerable discussion, among both physicians and surgeons, as to the propriety of surgical intervention in the severer forms of hæmatemesis. As I have operated upon six cases in which hæmatemesis was the only or the most prominent symptom, and as I have seen several similar cases in which I have advised against operation, my experience and the reasons for my action are, perhaps, worthy of record.

It is probable that the statistical tables and statements at our disposal, framed in different fashion by various writers, so frequently and confidently quoted are quite unreliable, and confusing rather than helpful. No clear distinction in any of these statistics seems to have been drawn between the hemorrhage from acute and from chronic ulcers, yet the distinction is one, it seems to me, of the most crucial importance. I propose, therefore, to ignore the statistical side of the question, and to deal solely with those facts which have been most vividly impressed upon me by the cases I have myself witnessed.

Hemorrhage may occur from acute and from chronic ulcers of the stomach, and whereas in the latter form the bleeding may vary within very wide limits, both as regards quantity and frequency, in the former the clinical history is repeated in case after case with the most remarkable monotony.

Hemorrhage from an Acute Ulcer.—When a severe attack of hæmatemesis occurs from an acute ulcer, it is but seldom that we can obtain a history of antecedent symptoms of gastric discomfort. Hemorrhage is in a large proportion of cases, in seventy-five per cent. according to Fenwick, the first symptom. If earlier symptoms can be elicited, on close inquiry they are found to have been very brief and trivial, and to have attracted no serious attention from the patient. The amount of blood lost is always large, and it is

lost within the space of a few minutes. A pint, a pint and a half, or even more, may be ejected in two or three rapidly succeeding efforts. The general symptoms are those attendant upon any serious loss of blood, collapse, more or less profound, blanching, sweating, rapid, shallow breathing, thin and quick pulse. The body-surface becomes cold, pallid, clammy; the patient is restless, and complains always and constantly of a thirst which cannot be quenched. If the bleeding ceases, reaction may soon be noticed; the temperature, which had been depressed, gradually rises and may reach 100° F. or 101° F., and color returns to the lips and cheek. Thirst and restlessness generally persist.

As a rule, the outburst of hemorrhage is not repeated, or, if repeated, is not severe. A mouthful of blood may be brought up once or twice in the course of twenty-four hours, but there is rarely more than this.

The rapidity of the outpouring of the blood may be gauged with seeming accuracy by noting the appearance of the vomit; bright fluid blood has escaped rapidly from the bleeding vessels, dark-brown blood, or fluid black as ink, has been long stagnant in the stomach and has been changed in color by the action of the gastric juice.

The characteristics, therefore, of the hemorrhage from an acute gastric ulcer are spontaneity, abruptness of onset, the rapid loss of a large quantity of blood, and the infrequency of a repetition of hemorrhage in anything but insignificant quantity.

Hemorrhage from a Chronic Ulcer.—In all cases in this group there have been symptoms of chronic indigestion, pain, or fulness after meals and occasional vomiting for months or even years, and there is constant complaint of greater discomfort in the later days. The bleeding varies within the widest limits of frequency and quantity, but, speaking roughly, we may say that the cases are divisible into two groups: In the one, the hemorrhage is trivial in amount, capricious in onset, irregularly repeated, and is, indeed, merely an unexpected, and, on the whole, an unimportant addition to the usual attacks of vomiting. From time to time a tinge of blood, or even a few ounces, may be observed in the food rejected in one of the constantly recurring attacks of vomiting. It is probable that small quantities of blood are not infrequently present in vomited matter or in the stools and pass unobserved, owing to the absence of symp-

toms. The estimates given by authors as to the frequency of hemorrhage in cases of chronic gastric ulcer vary between twenty per cent. and eighty per cent., and this difference is due not so much to the variations in the disease, as to the closeness and accuracy and continuity in the observations made with reference to the constituents of the stomach-contents and of the fæces.

In the second group the hemorrhage is the predominant feature. In a patient who has been subject for years to indigestion there is a sudden attack of hæmatemesis. This attack may or may not be, but in my experience generally is, preceded by a notable accession of pain and gastric irritability for, at the least, two or three days. The hemorrhage is copious, more than half a pint, and very commonly a pint, being lost. After a quiescent period of twenty-four hours or more a second equally severe attack occurs, to be followed in its turn by a period of quiet, which ends abruptly with a sudden and generally a more serious bleeding. In each attack the bleeding is free, but is rarely so copious and overwhelming as in acute ulceration. In rare cases, when a large vessel is opened by the ulceration, the hemorrhage may be inundating and cause the almost sudden death of the patient. Such a catastrophe is, however, very unusual. So far as the general effect upon the patient's condition is concerned, it is usually noticed that after hæmatemesis from an acute ulcer a speedy recovery is made. The pulse quickly regains its former good volume, and the effects of the depletion speedily pass off. In the hemorrhage from chronic ulcer a persisting anæmia is almost constantly observed, and in some cases is an obvious and the most striking feature of the case.

The characteristics, therefore, of the hemorrhage from a chronic ulcer, omitting the inconspicuous and the immediately fatal cases, are *the onset after a long history of digestive disturbances, culminating in acute discomfort for a few days, the tendency to recurrence with brief intermissions of a few hours or a day or two, the moderate quantity of blood ejected in each outbreak, and the condition of anæmia produced by the repeated loss of blood.*

It is mainly in regard to the treatment by operation of these forms of hemorrhage that differences of opinion have been expressed by physicians and surgeons who have written upon the subject. The most ardent advocate of the early adoption of surgical measures is Dieulafoy, who advises that even in a first attack of

hemorrhage an operation should be performed if half a litre of blood is lost. It seems to me that an approach to a successful solution of this most difficult problem of when to operate may best be made by accepting the distinction between hæmatemesis from an acute and hæmatemesis from a chronic ulcer, and by endeavoring to realize the exact pathological conditions which may be present in each of these varieties.

In those cases of hemorrhage from an acute ulcer in which an operation has been performed the conditions found have been differently described. In many, certainly in a majority, the blood has been observed to come from many simple erosions or superficial pittings on the mucous membrane. It is as though the membrane were quietly "weeping" blood. No spouting vessel has been observed, no single point from which the blood chiefly ran. An abundant oozing from many points is, rather, the type of bleeding seen. The large quantity of blood poured out is due to the multiplicity of the points from which it oozes, and not to the size of any single ulcer. In some a small, round, sharply defined ulcer, or two or more such ulcers, have been observed, and excision, or transfixion of the base of the ulcer, or ligature "en masse" has been performed. When multiple points of oozing have been found, such treatment has chiefly been impossible. Under such circumstances the cautery has been employed, or a styptic has been wiped over the surface, or temporary pressure has been made with gauze soaked in some astringent, or finally gastro-enterostomy has been performed.

The cases in which a definite bleeding-point has been seen and secured or the ulcer excised are certainly in a small minority. In the greater number of cases a general oozing has been observed, and some of the measures already enumerated have been adopted.

It is, perhaps, neither easy nor safe to judge of these cases merely by the reports which have been published, but, taking into account the characteristic features of the hemorrhage from acute ulcers, and especially the very marked tendency to spontaneous arrest of the bleeding, it is difficult to convince oneself that in these cases of "weeping" mucous membrane any real benefit has accrued from operation; the surgical interference seems rather to have been, in the successful cases, a complication in what would otherwise have been an uneventful recovery. The question is one, however, which cannot be settled with the knowledge at present at our command,

but I am inclined to think that in acute ulceration hæmatemesis does not offer much scope for satisfactory treatment by the surgeon. If, however, operation is for any reason deemed imperative, as, for example, when the hemorrhage is both copious *and recurring*, it is probable that a gastro-enterostomy, speedily performed, will be the most appropriate and most successful method.

In the hemorrhage from a chronic ulcer the case is far otherwise. In most of the recorded examples, an ulcer with densely thickened base and indurated edges has been more or less readily found; in many, the bleeding-point has been seen, and the ulcer excised or its base transfixed and the ligatures tied; in others, a gastro-enterostomy has been done with perfectly satisfactory results.

One can readily understand on examining a chronic ulcer which has bled intermittently how difficult it is for the hemorrhage to be checked spontaneously. The base of the ulcer is densely hard, and the vessel traverses it like a rigid pipe. One side of the vessel is destroyed by the ulceration, which makes a ragged hole therein. The vessel is unable, owing to the stiffening by chronic inflammatory deposit, to contract or retract, and the bleeding can, therefore, only be checked by the plugging of the opening by a thrombus, which may readily be detached or destroyed.

In one of the six cases below recorded, in which the hemorrhage had been excessive and frequently repeated, the ulcer was exposed at once; it was on the lesser curvature, towards the cardiac orifice. Excision was performed by making an elliptical incision round the base; when the first half of the incision had been made and the ulcerating surface exposed, the bleeding was readily seen. The wound resulting was stitched up and gastro-enterostomy was not performed. In view of the issue of this case, I much regret that I did not short-circuit. In the remaining five, one or more ulcers were found; in one, an ulcer on the posterior surface was excised and gastro-enterostomy performed to the opening left, and in four, a gastro-enterostomy alone was thought necessary. It is a striking fact, though possibly only a coincidence, that the only case in which gastro-enterostomy was not performed was the only case I lost. That the short-circuiting is of value in expediting the healing of an ulcer there can be no doubt, but in what way precisely it so acts is difficult to say. The more ready emptying of the stomach is

probably the chief and the most important result of the anastomosis, but the avoidance of, or the nullifying of the evil effects of, a pyloric spasm is certainly a factor.

To sum up, I would suggest that the treatment of hæmatemesis will depend upon the nature of the ulcer from which the blood is coming. In hemorrhage from an acute ulcer medical treatment alone will in almost every case suffice; surgical measures will only be necessary when the bleeding is copious and recurring. If any operation has to be done, gastro-enterostomy will probably prove the most effective.

In chronic ulcer operation should be advised as early as possible. If readily exposed, the ulcer, if solitary, may be excised, but a simple gastro-enterostomy is probably sufficient, in the great majority of instances, to secure the arrest of the hemorrhage and the rapid healing of the ulcer.

CASES OF HÆMATEMESIS DEALT WITH BY OPERATION.

Name and Date.	Sex.	Age.	Symptoms and Description.	Condition found ; Operation.	Result.	Remarks.
M. P. March 19, 1901.	Male.	30	Symptoms five or six years. Pain in epigastrium after meals ; most severe in bed at night ; slight pain between shoulders. Vomiting for twelve months, almost regularly on alternate days, "watery phlegm, very sour," once blood. Lost weight. Always careful in diet. Has attacks of faintness and prostration followed by melena. Diagnosed as "duodenal ulcer." Dilated stomach. He sought advice because of the unaccountable attacks of profound faintness and collapse which sent him to bed for two, three, or four days on many occasions. The melena which was then discovered was found afterwards to be always associated with the attacks. He is pallid, breathless, and very anæmic.	Ulcer in first portion of duodenum, with many adhesions. Small scar of gastric ulcer on posterior surface. Posterior gastro-enterostomy.	Recovered.	Dr. Millhouse. Dr. Anning. May 31, 1901. Gained twelve pounds. Has been quite well since the operation ; no gastric or duodenal symptoms. April, 1902.
M. C. April, 1901.	Male.	48	Under medical treatment five years with stomach disorder. Has been carefully dieted, and still is always expecting, and generally has, pain, distention, and uneasiness after food. Has bled freely two or three times recently. Stomach dilated. The symptoms since Christmas have been those of Reichmann's disease, hyperchlorhydria, etc. The recent occurrence of severe hemorrhage, necessitating rest for two or three days in bed, has alarmed him. Three attacks of hemorrhage in medium quantity during the last ten days.	Large ulcer, at least size of a florin, on the posterior surface near the pylorus. A scar of a smaller ulcer is seen on the posterior surface about three inches from the pylorus. Posterior gastro-enterostomy. Owing to stoutness and rigidity of muscles, I was hampered during the operation, and made a smaller opening than usual.	Recovered.	Dr. Ellis, Halifax. A ventral hernia in upper part of scar. A Fagenstacher thread, with which I stitched up the abdominal wall, suppurated and discharged itself. Quite well in August, 1902.
A. H. Reg. No. 3223.	Female.	24	Has had characteristic symptoms of gastric ulcer for about eighteen months. For the last six months has been kept in bed under medical	An ulcer close by pylorus with thickening over an area the size of a shilling. A dense,	Recovered.	Dr. Clarke, Wakefield. After leaving hos-

July 11, 1901.		treatment, but has vomited almost daily, and on all occasions blood has come. Is very pallid and anemic. In hospital she vomited daily fresh and partially digested blood. She is very much blanched, has attacks of fainting in bed, and looks very ill.		hard area with reddened surface and edges, and covered with lymph on the posterior surface. Posterior gastro-enterostomy. An ulcer, probably the bleeding one, on posterior surface being excised and the opening left united to the jejunum.	pital was sent to a convalescent hospital, where she remained for weeks; was discharged in good health, having gained nine pounds in weight.
N. G. January 9, 1901. (Cromer Terrace.)	Female. 26	For the last few months has had pain always after food, coming on immediately and lasting for three or four hours; has therefore limited her diet and has lost weight. Four weeks ago had hematemesis and melena. The melena has continued ever since, and is now threatening to end disastrously. Has fainted in bed several times. Pulse 96; very pallid. Has become much thinner during the last four weeks. During the three days before operation there were several motions daily, black in color and very offensive.	Recovered.	An ulcer in stomach on posterior wall. An ulcer elongated and thickened in duodenum, feeling like a date. Many adhesions. Posterior gastro-enterostomy.	Dr. Ellis, Halifax. This patient made a perfect recovery, and is now, September, 1902, in splendid health. She has regained all her lost weight.
M. P. July 10, 1902.	Male. 18	Four years ago had scarlet fever; since then has never been quite robust. Has suffered at times from pain after food and vomiting. These would be present for two to three days, then would abate, and he would be quite well for a few weeks. Has been careful in his diet. An ordinary hearty meal would invariably arouse the symptoms. Had no hematemesis. On July 6 ate an ordinary breakfast; late in the morning felt ill, epigastric pain, and vomiting. At first vomiting seizure only food came back; at the second, a "quart of blood." Later in the day vomited blood, a pint and a half, measured by his doctor. On the 7th vomited blood twice, on each occasion over a pint of clot.	Died.	The abdomen was opened through the right rectus muscle. Almost at once a hard patch about the size of a shilling was felt in the anterior wall of the stomach near the lesser curvature towards the cardiac end. This was the ulcer. An incision made by the side of this showed the ulcer in the act of bleeding. The ulcer was cut out by an elliptical incision enclosing it. The horizontal wound thereby resulted.	Dr. A. A. McNab. The mother of this patient had suffered from gastric ulcer when twenty-one years of age. Death occurred suddenly on the eighth day, during the administration of an enema. No apparent cause. The progress of the

CASES OF HÆMATEMESIS DEALT WITH BY OPERATION.—Concluded.

Name and Date.	Sex.	Age.	Symptoms and Description.	Condition found : Operation.	Result.	Remarks.
M. W. July 19, 1902. Reg. No. 3349.	Female.	24	<p>On the 8th twice, on the 9th three times, on the 10th once in large quantity and once in small. When I saw him he was very blanched, his face waxen and sweating. The pulse was 114, very thin and feeble. He had been free from abdominal pain since the attack of vomiting. The hemorrhage had told upon him severely. The persistence of symptoms of gastric ulcer for four years, with hæmatemesis of five days' duration, made the diagnosis chronic gastric ulcer, with recent deepening of the ulcer. It was probable, therefore, that the bleeding from a dense indurated area would not stop spontaneously, and I therefore advised operation.</p> <p>For fifteen months has had symptoms of gastric ulcer, pain, vomiting, and inability to take solid food. Eleven weeks ago the symptoms became more pronounced. Vomiting became frequent. During the last five weeks has vomited daily, and on almost all occasions blood has come. While waiting in hospital she vomited three times in five days, and on each occasion about half a pint of blood came away. She is profoundly anæmic, and looks very worn and ill. Basal hæmic murmur.</p>	<p>ing was closed transversely. A careful examination of the rest of the stomach was made, but nothing abnormal found. The abdomen was closed in the usual manner.</p> <p>Stomach moderately dilated. On the anterior surface near the pylorus were two scars equal in size to a three-pence piece, distant about one inch from each other. A few thin adhesions between these and the anterior abdominal wall. On the posterior surface near the pylorus an indurated area about one inch in diameter, with reddened area around it and covered by recent lymph, was found. Several adhesions to the transverse mesocolon. Posterior gastro-enterostomy.</p>	Recovered.	<p>case had been perfectly satisfactory. There had been no pain, no vomiting, no untoward symptoms; fluid had been taken in moderate quantity.</p> <p>Dr. Erskrigge, Royston. An excellent recovery; vomited once after operation, on sixth day, about four ounces of bile, no blood.</p>

FRIGHT AND DEATHS IN CHLOROFORM NARCOSIS; MENTAL PREOCCUPATION AS A PRELIMINARY TO GENERAL ANÆSTHESIA; RESECTION OF TUBERCULOUS TESTICLE.

**CLINICAL LECTURE DELIVERED AT THE NEW YORK POLYCLINIC HOSPITAL AND
SCHOOL FOR GRADUATES IN MEDICINE.**

BY J. A. BODINE, M.D.,

**Adjunct Professor of Surgery at the New York Polyclinic; Attending Surgeon
at St. John's Hospital, Long Island City, New York, etc.**

GENTLEMEN,—Chloroform possesses many advantages over ether as a general anæsthetic. Unfortunately, however, according to available statistics, the administration of chloroform has been followed by a considerably larger percentage of deaths from the anæsthetic than when ether was employed. It seems not improbable that this unfortunate fatality might be offset, to a very great extent, at least, by the deaths that take place, some time after operation, from kidney-irritation and various forms of lung-involvement, when ether has been the anæsthetic employed. The palsying discouragement, however, of a death on the table from the anæsthetic keeps many surgeons from the use of chloroform for general anæsthesia who would be very willing to employ it because of other recognized advantages.

When death takes place from chloroform, it is found invariably that the cause of death was a vasomotor paralysis. That is to say, the nervous system lost control over the motor nerve leading to the blood-vessels, and especially the veins of the body, and as a consequence the patient bled to death into his own tissue.

Now it is well known that deaths from fright occur in just the same way as deaths from chloroform poisoning. No pathological conditions are found in fatal cases of fright except a dilatation of the large veins of the body, and consequent marked passive venous congestion.

There is no doubt that many patients contemplate the taking of an anæsthetic with feelings of great fear. In a certain number of

cases this has even been sufficient of itself, without the administration of chloroform, to cause death. In one well-known case in a New York City hospital, the patient, a very nervous individual, became so worked up before the operation that the rhythm of his breathing was seriously disturbed, and the anæsthetist resolved to give him some training in deep breathing before he began the administration of the chloroform. Accordingly, when the patient was all ready for the anæsthetic, the cone was placed over his mouth, and he was instructed to breathe deeply. The instructions had to be repeated, because his nervousness interrupted his breathing. He seems to have believed that the anæsthetic was being administered, and after a few gasps he entirely ceased to breathe, and in spite of every effort could not be resuscitated. He had died from fright before the administration of a single drop of chloroform.

At the autopsy, nothing was found except the vasomotor paralysis and the consequent venous congestion, especially in the large veins of the abdomen, of which we have already spoken as common pathological results of fright and chloroform. Had the patient received even a whiff of chloroform, this death would surely have been set down in the records as due to the use of chloroform as an anæsthetic. The autopsy findings are exactly the same in both classes of cases, and even a few drops of chloroform are supposed to be sufficient to cause the vasomotor paralysis to which deaths from chloroform are uniformly attributed. As a matter of fact, it is practically always during the preliminary stage of anæsthesia from chloroform, and at a time when very little of the drug has been inhaled, that death takes place.

Another very interesting case, that shows how much influence fright may have on patients, occurred here at the Polyclinic Hospital not long ago. A young German was suffering from hemorrhoids of a sufficiently severe type to require operation. He was an intensely nervous individual and fearful of the result of the operation. His extremely neurotic condition had made it practically impossible for him to stand the pain from the hemorrhoids. After the operation was determined upon, he brooded over it very much, and remarked to patients in the wards that he should never survive it. He was in a state of nervous depression when placed upon the table. Before any of the anæsthetic was administered, an enema was given in order to secure preliminary cleansing of the rectum.

The patient thought that this was the beginning of the operative procedure, and in the midst of it ceased breathing, and though immediate and prolonged efforts were made by means of artificial respiration and stimulation to restore him, they proved unavailing. The autopsy showed perfectly healthy organs and nothing abnormal except the tense abdominal veins, in which nearly all the blood of the body had collected as the result of the vasomotor paralysis consequent upon the fright. In this, of course, we have another case in which had even the slightest amount of chloroform been given, another death from chloroform anæsthesia would have been added to the statistics.

In considering the statistics of death from chloroform anæsthesia, it is evident, then, that we must remember that fright and chloroform cause death in the same way, and that fright may also be an element in the production of death in cases where chloroform is used. As a matter of fact, seven out of every ten of the deaths reported from chloroform anæsthesia occur during the preliminary stage and at a time when the patient has inhaled only from a few drops to a drachm of the anæsthetic. If it were true that death in these cases was due to the toxic effects of the chloroform, then there would be some proportion between the amount of chloroform administered in each case and the number of deaths reported. As a matter of fact, this is not true, and in order to explain it, the existence of an idiosyncrasy for chloroform must be assumed. The three out of every ten deaths that take place after a considerable amount of chloroform has been administered are probably genuine examples of the true toxic effect of the anæsthetic. The other seven deaths are due to a combination of fright and chloroform, and sometimes the chloroform has so little to do with the fatal issue, so small an amount of the drug having been inhaled, that we cannot but think that fright is the most important or perhaps the only real factor in the fatal termination.

There is some negative evidence that serves to show this very well. It is well known that chloroform is the anæsthetic of choice in obstetric cases. No practitioner, as a rule, hesitates to give chloroform during the progress of labor. So few are the deaths at this time that the mortality of chloroform anæsthesia during labor is considered to be practically *nil*. The cause is not far to seek. Women are not fearful with regard to the administration of

an anæsthetic during labor, but, on the contrary, beg to have it administered. Though females are, in general, much more liable to serious nervous disturbance than men, their anxiety to be relieved of the pain makes them oblivious of any feeling of fright with regard to the anæsthetic. Hence the absence of mortality.

Chloroform is considered by most surgeons the anæsthetic of preference when children are to be operated upon. Children may be scared by the preparations for an operation, but they are not frightened, as adults are, and consequently do not suffer so readily from the fatal vasomotor paralysis that causes death in chloroform anæsthesia. The same thing is true of the negro race in our Southern States. The negro, as is well known, stands chloroform very well, and there are many practitioners in the South who, from their experience with this race, are sure that there cannot be nearly so much danger in chloroform anæsthesia as would seem to be announced by the statistics gathered from more highly cultivated classes of people. As a matter of fact, while the negro dreads surgery very much, he has a childlike confidence in his physician. He says to himself, "Dr. So-and-so would surely do nothing to hurt me. He has told me there is no danger of my dying." As a consequence, deep-seated, fright-involving disturbance of the nervous mechanism that rules over the vasomotor mechanism is not so apt to be present in the negro, as in more intelligent patients. The consequence is the lessened death-rate from chloroform-anæsthesia among the colored race, not because negroes are so much less susceptible to chloroform, but because their fright is not the serious auxiliary condition that it so often is in white patients of the more intelligent classes.

The negro may be killed by fright quite as well as the white man. A good illustrative example of that fact is said to have occurred in one of our large Western universities. A number of medical students, knowing the dread that negroes have of the dissecting-room, and that they have never entirely abandoned the tradition that at times negroes are captured by the students in order that their bodies may be used for dissection purposes, pounced upon a negro who was slinking past the anatomical department, and, having covered his head with a bag, succeeded in carrying him to the dissecting-room. His face was uncovered, and he was allowed to see the subjects prepared for dissection before he himself was

laid on the table. His face was covered once more, and then, his body having been uncovered, one of the students remarked that he would open the abdomen. Presumably for this purpose an icicle was drawn in a single line down the abdomen of the intensely frightened negro. He struggled a little, gasped a few times, and then stopped breathing. Every effort at resuscitation proved unavailing. He died from fright.

This case illustrates how much fright may mean as an auxiliary to the chloroform toxæmia in the production of a fatal issue in these cases. That the negroes do not suffer more from chloroform anæsthesia they owe, then, to their childlike confidence in their physicians, and the consequent suppression of this fatal accessory to the direct effect of the anæsthetic.

Before administering an anæsthetic, then, it is important, first of all, that the patient's fright should, as far as possible, be eliminated. He is under the control of a dominant idea that makes the ordinary regulating mechanism of his vascular system especially prone to be ineffective. This dominant idea can be gotten rid of only if some other idea is made to take its place by giving the patient another mental preoccupation. I think that I have demonstrated that this can be accomplished with much less difficulty than might be imagined. Of course, it is important that the anæsthetist or the attending surgeon should have the complete confidence of the patient. It may be necessary, too, that the surgeon to whom the patient came originally, and on whom he depends for the success of the operation, should be present at the beginning of the administration of the anæsthetic. If this precaution is taken, however, and the patient is treated in the method that I shall describe, not only is all danger of fright eliminated, but most of the annoyance of beginning anæsthesia is made to disappear, and the patient comes under the influence of the anæsthetic without any of the serious annoyance for the anæsthetist or the patient himself that is so frequently experienced during the first few minutes of the administration of the anæsthetic.

It might seem that any method that would accomplish this would be so difficult of application as to make it impossible in general use. As a matter of fact, as you will see here in my ward in the Polyclinic, hospital-residents readily learn to direct patients and give them that mental preoccupation which saves them from

fright and does away with the struggles at the beginning of anæsthesia. Our patient for this morning is suffering from tuberculous enlargement of the testicle, and besides being a nervous individual, his general health is not very good, and his anxiety with regard to the effect of an anæsthetic is excessive. I think, then, that this will be a fair case on which to illustrate for you the possibilities of the method which I have found so useful.

PRELIMINARY SUGGESTION IN GENERAL ANÆSTHESIA.

I am going to show you now, gentlemen, that it is perfectly possible to bring a patient under the influence of a general anæsthetic without the necessity for employing the slightest amount of force in holding the anæsthetic to his face, or keeping his hands from disturbing the anæsthetist, or even changing his position on the table. You know, of course, the old proverb, "Pet dogs and little children and chemical experiments never show off well in company." It may prove that my expectations will be disappointed because of an especially susceptible and nervous patient. The patient has not been selected for the occasion, but is just the next one on the list for operation. I should be perfectly sure, if I had known him for a couple of days, that I would have influence enough over him to prevent any display of uneasiness or any disinclination to the anæsthetic during its administration.

I tell him now to put his hands tightly together, the fingers interlacing, and to grip them firmly. I ask him to fix his mind on that action, assuring him that the tighter he grips his hands, the sooner and easier he will go under the influence of the anæsthetic. I ask him to listen for my voice and to do what I tell him, no matter what other thought may come into his mind. As I give him the first whiff of the chloroform, I remind him once more to keep his hands firmly together and then direct him to breathe deeply and quietly, not minding the sensations that come over him. I talk to him quietly, and assure him every little while that everything is all right, and that his hands must be kept together. If you were to feel his pulse, you would find that it did not vary in number or quality from what it was before he began the inhalation of the anæsthetic. I keep reminding him every little while to breathe deeply, and occasionally I recall to him that he must grip his hands firmly together.

If you have been paying attention, you will have noticed a short stage of slight tremor and almost excitement that passed over him. There was, for a moment, a tendency to lift his legs and to throw his hands about. Before he had separated them, however, my reminder to him that everything was all right, and that his hands should be kept together, restrained him. After a few moments I shall have a word to say about this stage of chloroform narcosis through which he has just passed.

There is nothing to do now except to remind him once more to breathe deeply and to continue speaking reassuringly to him. At a moment when sensibility is greatly reduced, and when a condition of primary relaxation has been obtained from the anæsthetic, patients are, as a rule, still quite conscious of their surroundings. During this time it is important not to say anything that will disturb the patient's equanimity. General conversation between surgeon and assistants must not be permitted, and special care must be taken to avoid the discussion of any surgical doubts or difficulties or recent fatalities that might seem to the patient to portend a serious result in his own case.

A recent happening here in the Polyclinic Hospital will illustrate very well the danger of neglecting this precaution. Just as one of our patients was entering the stage of narcosis, when all his inhibition was for the moment suspended, but consciousness was not quite lost, a convalescent from one of the wards was leaving the hospital, and in getting on the elevator bade good-by to the nurse. In womanly fashion a number of good-byes were said. The patient's pulse, that had been very quiet just before, running not more than ninety to the minute, jumped suddenly to 140 and then 160. A state of almost fatal collapse ensued. Constant repetition of the word "good-by," heard plainly in his semi-conscious condition, was translated by his subliminal consciousness into a direct allusion to his own condition. It is easy to understand how serious might be the result under such circumstances.

I am firmly of the opinion myself that patients who die in the primary stage of chloroform anæsthesia really die from fright. It must be remembered that most people who die from chloroform, die at the very beginning of the administration of the anæsthetic, at a time when usually considerably less than a drachm of the drug has been inhaled. They do not die from the toxic effects of the

chloroform. There is some other reason, and this evidently psychic in character. Deaths from fright, after all, are not very uncommon. If a husband's dead body is brought suddenly in to a wife, she may drop dead from excess of emotion, even though no serious pathological condition is present in any of her important organs.

It is a matter of statistics that more people die in train-accidents that happen during the day than during the night. When a train goes over a bridge, or a bad collision takes place in day-time, the passengers have had some little warning of the catastrophe, and even slight physical injuries may cause death. On the other hand, very serious injuries received at night and without warning are often recovered from, contrary to all expectations.

I pointed out to you the transition-stage in the development of the anæsthesia when our patient, notwithstanding my constant suggestion, made some slight movements and seemed in a state of excitement. This represents the moment when inhibition—that is, the faculty of self-control and of control of the movements of the body—is dissolved. Consciousness continues after this, but the result of any sudden emotion is an intense nervous reflex, because of the total absence of inhibition in this peculiar subconscious state.

Surgeons who make a habit of talking about the difficulties of a prospective operation, or who, during the development of anæsthesia, even hint at the possibility of a fatal termination in the case, always make an unfortunate mistake and one that may have serious consequences. As a rule, the greatest care should be exercised with regard to what is said, even when the patient seems to be fully under the influence of the anæsthetic. A number of cases have been reported in which, though patients suffered not the slightest pain, and seemed to be completely under the influence of ether or chloroform, they were still able to understand thoroughly and recall very accurately all that was said or done during their supposed state of unconsciousness. Quite often consciousness is not completely lost, even in the midst of perfect anæsthesia. This fact must be carefully borne in mind.

You see, then, that our patient has been brought under the influence of the anæsthetic without a struggle. If chloroform were always given in this way, there would not only be many less deaths, but there would also be very little trouble in its administra-

tion. Make it a rule never to use force of any kind when chloroform is being given. Give the patient a preoccupying thought, such as the gripping of his hands, and the constant reassurance of your voice, and there will usually be no inconvenience either for you or for him.

With regard to the use of ether or chloroform as general anæsthetics, I personally prefer chloroform. If the deaths from fright that occur during the administration of chloroform were eliminated, then it would be a much safer anæsthetic than ether. When chloroform is an enemy, it is always open and above-board. It kills at once and directly. Ether is an insidious foe. The patient may survive the operation, but dies from irritation of his lungs or of his kidneys ten days after the operation. If I had to choose an anæsthetic for myself to-morrow, I should take chloroform, but I should want it administered by a careful expert anæsthetist. Under such circumstances it is doubtful if more than a modicum of the deaths that are now reported from chloroform anæsthesia would occur.

RESECTION OF TUBERCULOUS TESTICLE.

The patient whom we have anæsthetized so exemplarily is a young man of twenty-two, who suffered from severe Pott's disease four or five years ago and has a marked kyphosis in his lower dorsal region. He came to the dispensary about a year ago because of some discomfort in his scrotum. Examination showed the presence of some nodules in his epididymis. Under the circumstances the suspicion was at once aroused that these were tuberculous in character. Constitutional treatment was employed, and his general health was improved as far as possible. Applications of ichthyol were made to his scrotum, but all to no purpose. Six or seven months after he first came under observation, some irregular nodules could be felt in the other epididymis. No doubt of the diagnosis could any longer be entertained, and the patient was advised to have the tuberculous tissue removed. Castration was not suggested, however.

I should not consider myself any more justified in taking out these testicles because of the tuberculous nodules in the epididymis than I would of removing ovaries because of the presence of small cysts in them. One operation is as little justified as the other. What we shall do here is to remove the epididymis and leave the testicle. Even if we should have to remove the entire epididymis.

it is wise to leave as much healthy testicular tissue as possible. Even though the testicular portion left should be quite small in amount, it will, as in the case of the ovary, produce its physiological and sexual effect upon the man, and though he remain sterile, he will not be unsexed.

In order to remove the diseased tissue, our field of operation must be as free from blood as possible. To effect this, we will employ a bloodless method, which you will find in none of the text-books, but which is worth knowing. I grasp the scrotum along the raphe between the testicles, and pass a needle with a ligature through the tissues. The ends of the ligature are carried around that side of the scrotum which one wishes to have bloodless. The pressure upon the artery of the cord is sufficient practically to stop the circulation. Beneath the ligature here on the outside of the scrotum we place a small wad of cotton, partly in order to save the delicate skin, and partly in order to prevent too great pressure on the cord. In a case like the present, it makes no difference whether the vas deferens is pressed upon or injured or not, since after the removal of the epididymis it will lose its function. In any case, however, the insertion of the wad of cotton beneath the ligature prevents any lasting injury, even to the vas.

In removing the epididymis the rule is to keep close to the globus major of the epididymis, and permit all traumatism in the operation to occur at the expense of epididymal structure. The testicle itself must be absolutely spared, unless there are nodules of disease in it. You can see during the course of the operation how well our improvised tourniquet produces the desired effect. There is scarcely any hemorrhage to obscure the field of operation, and we could not effectively guarantee ourselves against injury of the testicle if there were any bleeding. We shall now do the same operation on the other side. We shall leave the man his testicles, and though their blood-supply through the cord is cut off, they will retain their vitality because of the development of a collateral circulation from the scrotum after adhesions have formed. Thousands of testicles have been removed in such cases without any proper justification. The human testicle itself, though without its epididymis, is much better, even from a cosmetic stand-point, than any prosthetic apparatus, such as the celluloid or gutta-percha testicles that French specialists have suggested.

After we release the ligatures, we shall have some oozing of blood as the result of vasomotor reaction after the period of bloodlessness. This effused blood must be carefully removed, so that none of it is left to coagulate within the tunica vaginalis, for this would provide an especially favorable culture-medium for micro-organisms. If the oozing does not stop after a few minutes, as in the present case, some quite hot water should be employed. Do not trust to sensation, however, with regard to the heat of the water. Test the water with a thermometer. Testicular tissues are delicate, and you do not want to parboil them.

A practical point in the performance of these operations in the scrotal region, that you will notice we have employed in both the cases this morning, is this piece of rubber tissue enveloping the penis. It is impossible to make the urinary meatus aseptic. It is also impossible so to cover it with a gauze-bandage or towel that it will surely not escape from the covering and prove a source of infection in the field of operation. If a piece of thin rubber tissue be taken and rubbed with chloroform, it can be wrapped around the penis, and will cling to it until removed at the end of the operation. This precludes all danger of septic infection from this source. Many a suppurating hernia would be saved if this were a common practice, and the manipulation is so easy that it should be much more generally applied than it is.

A CASE OF MELOPLASTY; TUBERCULOUS GLANDS OF THE NECK.

A CLINICAL LECTURE AT THE POST-GRADUATE MEDICAL SCHOOL.

BY DANIEL N. EISENDRATH, M.D.,

Professor of Clinical Anatomy, College of Physicians and Surgeons (Medical Department of University of Illinois); Professor of Surgery, Post-Graduate Medical School; Attending Pathologist, Michael Reese Hospital, etc., Chicago.

A CASE OF MELOPLASTY.

GENTLEMEN,—This patient, aged thirty-five years, wants a plastic operation for a large deformity arising from the removal of a buccal tumor two years ago. In looking at him you will observe (Fig. 1) that the angle of the mouth on the left side droops; that there is a scar extending from the malar bone to the lower border of the lower jaw and from the angle of the mouth to the parotid region. The scar does not involve the entire thickness of the cheek, and, as you will note, the mucous membrane of the mouth has remained intact. There is great probability, therefore, that Steno's duct, which, as you will remember, opens opposite the second upper molar tooth, has not been injured. There has, however, been extensive destruction of the branches of the facial nerve which supply the chief muscles of expression, whose function is to raise the angle of the mouth and to depress the same. We cannot hope, therefore, on account of this extensive destruction, to obtain as good a functional result as could be wished for. The chief object which he desires is to be rid of a rather hideous scar. I propose, therefore, to excise the scar-tissue completely, through an incision made in the healthy tissues outside of the cicatrized area, and to fill in this denuded surface with a large flap which I shall slide upward from the side of the neck. The chief point about all of these plastic operations, especially if the flap is a large one, is to remember to secure the pedicle of the flap as close as possible to some large artery. For example, in making a rhinoplasty we attempt to shape the pedicle in such a manner that the terminations of the facial



FIG. 1.—Appearance of patient previous to a plastic operation for deformity resulting from the removal of a tumor of the cheek.

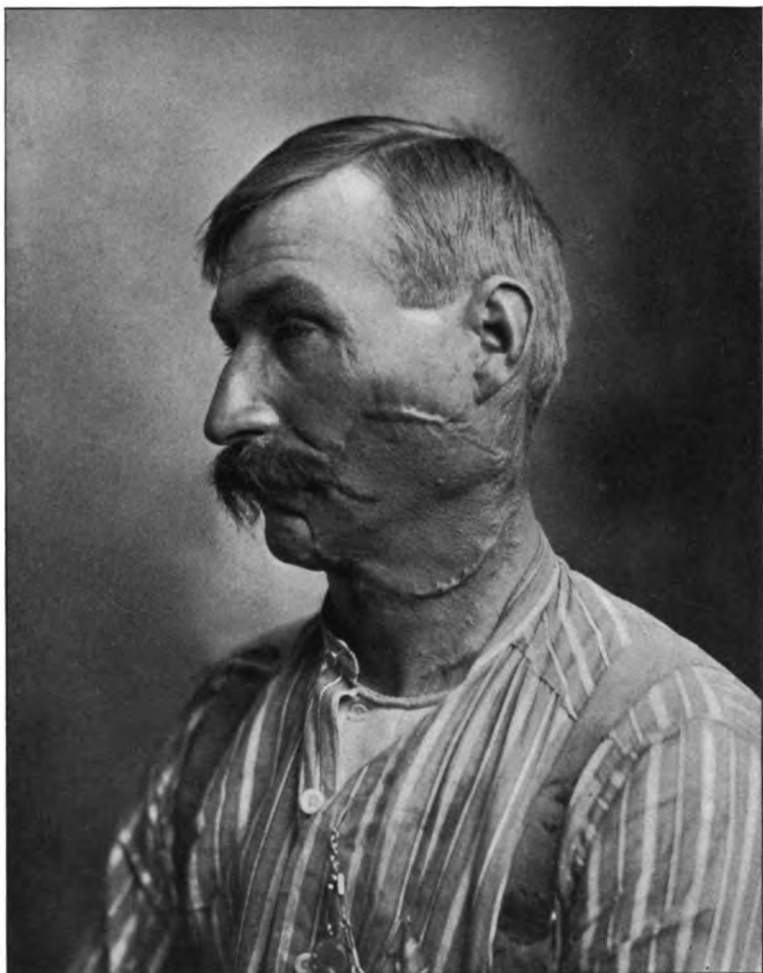


FIG. 2.—Appearance of patient after the operation.

artery shall supply the blood for the same, and in this case it will be my effort to have the pedicle situated posteriorly over the sternocleidomastoid, in such a manner that it will be well nourished by one of the branches of the carotid. It will scarcely be necessary to remark here that no tissue of the body is so well adapted to plastic operations as that of the face and neck, on account of the great elasticity of the skin. When a flap is taken from the adjacent portion of the body, as will be done in this case, and engrafted upon the previously denuded area, the operation is known as an autoplasty. When we take the flap from some distant portion of the body, as, for example, the arm, the operation is called a heteroplasty.

The first step in any plastic operation of this kind is to take a piece of gauze and lay it upon the surface to be covered, and cut out a pattern, so to speak, a little larger than the cicatrix. This will allow for the shrinkage which invariably follows after the flap has been severed from its surroundings. The gauze is then placed upon the surface from which we desire to take our flap (the side of the neck in this case), and with a sharp scalpel a very superficial incision is made along the edges of the gauze, which will give one the size of the flap.

I shall now proceed, after having prepared the parts in the ordinary manner, to excise the scar-tissue, and, having done this, shall follow the line in the neck which I have previously made with a scalpel around my gauze pattern. Having done this, you will notice that the flap fits quite accurately into the denuded area. I am extremely careful at this point not to rotate the flap more than 180° . A greater amount of rotation would interfere with the blood supply of the flap and cause gangrene of the same. As you will now see, I proceed to unite the flap with the edges of the denuded area, being careful to avoid any tension of the edges, and also securing very accurate approximation of the same. This can best be done in wounds of the face or neck by alternate sutures of the finest silkworm gut and horsehair, the latter material being exceedingly well adapted for accurate coaptation of the edges without danger of necrosis. The silkworm gut which I employ is specially selected, and much finer than that which is ordinarily used in laparotomies, etc. The surface on the neck, from which the flap was taken, is about three by four inches. I shall proceed to loosen up the edges

before uniting the same with silkworm gut and horsehair, which can be readily done on account of the great elasticity of the skin.

In some cases of meloplasty, especially those following noma, or cancrum oris in children, the scar involves the entire thickness of the cheek; and in such cases it is best to take two flaps from the neck, so as to cause the skin surface of one to be turned inward to the mouth, similar to the Thiersch operation for hypospadias, and the other to cover this in the ordinary manner of a plastic flap, so that the two raw surfaces are in apposition. The operation of turning the skin inward is to compensate for the loss of mucous membrane, the skin soon changing its character and assuming that of the mucous membrane.

[NOTE.—A week later the same patient was shown to the class. There had been primary union throughout, with the exception of a small surface at the lower portion of the neck. The drooping of the lip could not be entirely corrected on account of the absence of some of the fibres of the facial nerve. The man was advised to raise a moustache, and his appearance later on is seen in Fig. 2.]

TUBERCULOUS GLANDS OF THE NECK.

The next patient whom I present to you is the young lady upon whom we operated last Saturday for a tuberculous lymphadenitis of the cervical glands.

There are two sets of cervical glands,—the superficial and the deep. The superficial lie chiefly on the external surface of the sternocleidomastoid, and are less frequently affected in tuberculous processes. The deeper glands lie in four different places. First, contiguous to and frequently within the capsule of the parotid gland. Second, in the same relation to the submaxillary salivary glands. Third, along the carotid sheath, in which situation they are not infrequently very firmly adherent to the internal jugular vein, and the fourth situation, in the subclavian or posterior triangle of the neck.

Before speaking of the technic of operations for tuberculous glands, which holds true for glands which are inflamed from any cause whatever, I desire to speak briefly of the etiology, pathology, diagnosis, and prognosis.

I believe that we too often remove tuberculous glands of the neck without considering the fact that, with but few exceptions, it may

be stated as an axiom that an enlargement of the lymphatic glands is seldom primary; and we should make it a rule not to remove the lymphatic glands unless we are positive that the primary cause has been discovered and eradicated. To illustrate: I see a great many cases of tuberculous glands of the neck which have been operated upon, especially in children, in which there is a recurrence of fully as large a number of glands. There is the same tendency towards caseation and breaking down as at the operation performed, say six months or a year ago. The reason for this is that, in the majority of cases, the primary focus is situated either in the nasal or pharyngeal mucous membrane, or in the so-called pharyngeal tonsil, and most frequently in the faucial tonsil. A chronic rhinitis in children is frequently overlooked as the infection atrium. Every one sees cases where adenoids and caseous tonsils, greatly hypertrophied, their follicles filled with a cheesy detritus, are the atrium of infection for the tubercle bacillus. Now, these structures are all drained by the deep set of cervical lymph glands. It has been proven microscopically, through sections of these atria, that the tubercle bacilli get into the lymphatic system through these structures. If we operate upon such patients for tuberculous glands of the neck, without removing these primary foci, it would be far better for our reputation to have left them alone. They are almost certain to recur. I make it a rule to examine the nose, the pharynx, and fauces carefully in every individual afflicted with tuberculous glands of the neck, and remove adenoids and hypertrophied tonsils as a matter of routine practice in such cases.

The frequency of infection of the tonsils with tuberculosis has been explained by the fact that children, in creeping about the floor, are very apt to inhale the finely pulverized tuberculous sputum which results from the drying up of the expectoration of consumptives. Carious teeth are also a frequent atrium of infection, and these should be carefully inspected, especially in younger persons. Tuberculosis of the lymphatic glands is more frequent in the cervical group than in that of any other group of glands in the body, the order of frequency being, after the cervical, the axillary and inguinal.

In regard to the pathology, I have examined specimens which illustrate every stage of the morbid process. The tubercle bacillus is taken up by the gland which acts as a sort of filter. It is arrested

in the meshes of the same, surrounded by inflammatory cells, and a contest ensues between the cells of the lymph gland and the organism. If the former are successful, the focus becomes organized just like a tuberculous nodule in the lung, and scar-tissue replaces the former fine reticulum. We have glands oftentimes left in this condition, after the healing of a tuberculosis, chronically enlarged and firm. Should the organisms, however, be the victor, caseation takes place, either in one or in a number of places, within the glands, as in the glands recently removed. Many of the glands show five or six separate foci of caseation. Sooner or later these foci coalesce, and the gland is inverted into a mere capsule containing a soft cheesy material. On section, some do not even show caseous foci; but microscopically these same glands show typical tuberculous granulation-tissue and tuberculosis. At the same time that this process is going on inside of the gland, a rather important change takes place in the capsule and in the periglandular tissue. The latter become hyperæmic and undergo inflammatory changes, so that a periadenitis results. It causes adhesion of the glands to each other, to the vessels, especially to the internal jugular vein, to whose sheath they are frequently so firmly adherent that they cannot be separated without tearing the vein-wall. They also become adherent to the surface, permitting nature to discharge the contents of the gland spontaneously through the skin, but giving rise to rather unsightly sinuses, with exuberant tuberculous granulation-tissue, discharging pus from time to time. This periadenitis, as you noticed last week, is quite an important factor in the technic, and renders our operations much more difficult than if it were not present. In this manner you will have noticed how closely adherent one of the glands was to the spinal accessory nerve, the latter being almost buried in the adhesions.

In regard to the diagnosis, we may divide all enlargements of lymph glands into the acute and chronic. Of the former, the history and local appearance will aid in the diagnosis. We may have acute suppurations of the lymphatic glands following the various infectious diseases, especially scarlatina, and occasionally influenza. Perhaps the most frequent causes of acute suppurations of the glands of the neck are the infections following carious teeth in children, especially in the poorer classes.

In regard to the chronic enlargement of the glands, the most

frequent causes are tuberculosis and syphilis. The former constitute over ninety per cent. of all of the causes of glandular enlargements in the neck. In labial or mouth chancres the enlarged glands are often the first symptom which the patient notices. In all of these instances it is possible, as I have stated before, to find a primary focus. Among the glandular enlargements in which there is no primary focus, which are of a chronic nature, the most frequent are those of Hodgkin's disease or pseudoleukæmia, in which the glandular enlargement is usually symmetrical. The glands themselves are very large, occurring in groups, and seldom present in the neck alone. The glandular enlargements in lymphatic leukæmia, of which I showed you a case some months ago, are also symmetrical, as a rule. In addition to these primary and secondary enlargements, we must not forget that patients present themselves, from time to time, with enlargements of the cervical glands, which are apparently primary, but where a careful examination will reveal a focus of carcinoma either upon the lips, buccal mucous membrane, or, occasionally, in the sinus pyriformis close to the entrance to the larynx. We should, therefore, be extremely conservative about making a diagnosis of primary enlargement of the lymphatic glands, first exhausting every possible method to find a primary cause. It is of importance, in making a diagnosis of tuberculous glands of the neck, after examining the nose, throat, and ear, to make careful inquiry into the patient's family history, in order to ascertain whether tuberculosis in some form has not been present in it.

Just a word concerning the prognosis of tuberculous glands of the neck. It has been found that in twenty-five per cent. of the cases, tuberculous glands of the neck are followed by pulmonary tuberculosis. This, as you know, can take place, either as a portion of a general miliary tuberculosis through rupture of the gland into one of the larger veins, or by direct lymphatic connection with the apex of the pleural cavity. I think that one should not be too confident (1) about the non-recurrence of such glands after operation, and (2) that they may not be followed at some future time, even after a period of years, by a tuberculous focus either in the lungs, bones, or some other viscus. I say that one should be guarded in regard to the prognosis about recurrence, because, if the primary focus or atrium of infection in the nose, the pharynx, and occasionally in the middle ear or mastoid, is not removed, the patient will

come back very much disappointed within six months or a year, with almost as many glands as were removed originally.

The indications for operation are: (1) If a sinus is present, communicating with a gland from which pus is discharged from time to time, leaving a rather hideous and unsightly wound in the neck; (2) whenever the glands have increased rapidly within a short period of time; and (3) whenever the family history is such that pulmonary or any form of so-called surgical tuberculosis is to be feared, if allowed to remain.

I shall recall the exact technic which I employed in removing a large number of glands last Saturday. I prefer a straight incision made over the middle of the sternocleidomastoid muscle. One should never make the incision so small that it is necessary to work in the dark. Under such circumstances, it is almost impossible to avoid injuring important vessels and nerves, and also to avoid overlooking glands buried deeply in inflammatory tissue. In the ordinary case I make an incision from the angle of the jaw downward, its length varying with the number of palpable glands, but always making the external incision one to two inches below the lowest one to be felt. After dividing the skin and superficial fascia and exposing the sternocleidomastoid muscle, I then search for the anterior border of the latter, and, after exposing it, discontinue the use of the scalpel. If the glands are located chiefly under the axilla and along its anterior border, the muscle and skin should be retracted, so that the glands are fully exposed. As you will remember, from this point on I used a dull-pointed curved scissors and a retractor, with two sharp short points; the latter is hooked on to the bunch of the glands, which I then pull outward, and begin to snip the periglandular tissue with scissors. I am careful to keep the points of the scissors (which are dull) towards the gland, so that, if I cut any structure, it is the capsule of the gland, and never any adjacent blood-vessel or nerve. This strong traction or drawing away of the glands from the surrounding tissues enables one to determine perfectly their relations to such large vessels as the carotid artery and internal jugular vein, and in a series of one hundred operations of this kind, I have always avoided injuring either vessel by the use of this method, which I saw, for the first time, employed by Professor D. A. K. Steele, of this college. Having dissected away the glands in this manner from the carotid artery under the

anterior border of the sternocleidomastoid, I proceeded to remove the glands lying along and under the posterior border of the same muscle in a similar manner.

The main points in the technic to be remembered along this posterior border are: (1) that the capsules of the glands are frequently so intimately adherent to the sheath of the internal jugular vein that it seems almost impossible to avoid injuring the same. I believe that you can escape this unpleasant complication by using the method of pulling the glands away from the vein and using a dull-pointed curved scissors in the manner I have just described. In addition to the internal jugular vein, you will remember that I exposed the vagus nerve lying at a more posterior level between the carotid artery and jugular vein. Another structure, which I showed you and avoided carefully, was the spinal accessory nerve. This nerve passes through the substance of the sternocleidomastoid in a rather oblique manner, emerging at the middle of its posterior border, and passing across the posterior triangle of the neck to enter the trapezius muscle at the level of the seventh cervical vertebra. If this nerve is severed, the patient will not be able to raise the arm above the level of the shoulder, chiefly owing to paralysis of the trapezius muscle. You will remember that from the time I began to work along the posterior border of the sternomastoid I kept this nerve constantly in mind, and, although it was embedded in periglandular adhesions, we were able to expose it fully and to demonstrate its intact condition, by compressing it slightly with the tip of the scissors and noting the jerkings of the trapezius muscle. It is almost impossible to avoid injuring some of the superficial cervical nerves which turn around the posterior edge of the sternocleidomastoid muscle at about its middle, and their destruction will be followed only by some anæsthesia of the skin of the neck. Towards the latter portion of the operation, you will recall that I was obliged to expose the brachial plexus of nerves as they lay deeply in the posterior triangle, and also the apex of the pleural cavity. It is very easy to avoid injuring either of these structures, but not the rather fine cord which I exposed in this area on the left side of the neck, the thoracic duct. This duct has been injured a number of times during these operations for the removal of glands of the neck, and some twenty cases, I believe, were recently collected, including two of their own, by Drs. Plummer and Schroeder. The chief

result of cutting this duct is the constant escape of lymph from the wound for many weeks after the operation. The prognosis, however, even if it is cut, is good, as the opening closes gradually. Working deeply also in this supraclavicular region, I called your attention to the presence of the subclavian vein and artery. The thoracic duct curves around the lower end or insertion of the sternocleidomastoid muscle into the clavicle, and empties into the subclavian vein close to its junction with the internal jugular. These are among the most important anatomical points to be remembered during the operation. I feel certain that you can avoid injuring any of these structures by discarding the scalpel as soon as the sternocleidomastoid muscle has been exposed. The Kocher dissector is used by many, but one can accomplish almost the same results with the use of the above two instruments and the index-finger, the latter being used occasionally to loosen glands from their surroundings.

There is danger of injuring the facial nerve in operations around the parotid region. It curves around the lower end of the ear at a deep level, entering the parotid gland about opposite the tragus, and then emerging from it again and sending its branches through the anterior border of the gland. One is apt to injure this nerve, especially in removing glands situated deeply between the angle of the jaw and the mastoid, when the ear is pulled up and the relations are disturbed. Should it occur that the internal jugular vein has been incised or the carotid artery lacerated, the utmost presence of mind is necessary. Blind thrusting in of artery-forceps will not avail. The wound must be fully exposed after temporary tamponade, and then forceps placed upon the exact point. It will not be necessary, especially in the case of the vein, to apply more than a parietal ligature, unless great carelessness has been displayed in the performance of the operation, as the wound is usually in one wall.

The glands having been removed, a large wound surface is left, which, in the majority of cases, has been in contact with caseous material issuing from glands which have been ruptured during operation. It is a good plan to swab this whole area with tincture of iodine, or to dust it with iodoform. Before suturing the wound, you will have noticed that I tamponed every crevice of the area with plain sterilized gauze, preferring this to iodoform gauze, on

account of its better drainage properties. This has been experimentally determined. This gauze is packed in rather firmly and allowed to escape from the lower angle of the incision. It was almost completely removed upon the second day, the remainder being gradually withdrawn during the first week. The purpose of this gauze is twofold. First, it serves as a tampon to check the parenchymatous oozing from the raw wound surface, which is quite extensive during the first forty-eight hours. Secondly, it serves as a drain for the unavoidable suppuration of a tuberculous nature on account of the infection of the surface in the majority of advanced cases.

By examining the patient, to-day, you will note the ideal primary union throughout the wound, with the exception of the lower portion where the gauze protruded. The suture material used for this purpose was alternate sutures of silkworm gut of a rather fine quality and horsehair. The latter acts as an approximation suture of an ideal nature, and in using it one must not neglect to employ very fine cutting needles and to insert the sutures close to the edges of the wound. The silkworm-gut sutures are inserted about one-quarter of an inch from the edges of the wound. The entire incision, after being sutured, is sealed with collodion, extending as far as the gauze at the lower angle of the wound. In addition to the local treatment, one must not neglect the administration of tonics and to encourage the patient to live an out-door life after the operation.

**ARTIFICIAL RESPIRATION AND HEART MASSAGE
IN NARCOSIS; VOLKMANN'S SUSPENSION APPA-
RATUS; LIGATURE OF THE COMMON CAROTID
ARTERY; OPENING OF THE FRONTAL SINUS; RE-
SECTION OF THE SUPERIOR AND INFERIOR
ORBITAL NERVES; SUTURING OF THE BOWEL.**

BY EDMUND LESER, M.D.,

Professor of Surgery at the University of Halle.

GENTLEMEN,—I desire to impress upon you the fact that in narcosis the danger to life arises both from the heart and from the lungs. It is, therefore, of the utmost importance that not only the breathing be free, easy, and regular in asphyxia and syncope, but that the movements of the heart themselves are excited by proper stimulation. For these purposes the patient is placed perfectly flat on his back on the operating table, while the physician takes his place at the head of the table, grasps the arms at the elbow, while an assistant, if present, draws out the tongue and supports the lower jaw. By means of strong elevation of the arms above the head, the thorax is widened and the act of inspiration performed. After the arms have been held in this position from three to five seconds, they are quickly returned to the sides and the thoracic walls forcibly pressed in so as to drive out the air. If this procedure be properly performed, the sound of the air, as it passes in and out of the glottis, can be plainly heard. (Figs. 1 and 2.) Nélaton's pressure method may have to be practised or forced respiration with the bellows as recommended by Wood.

König has directed attention to an additional procedure of great value, namely, heart massage, which can be performed along with artificial respiration. This is done by making a rotary motion with the fist directly over the apex of the heart. The hypodermatic use of strychnine is also to be recommended, while electricity, nitrite of amyl, suprarenal extract, venesection, rhythmical traction

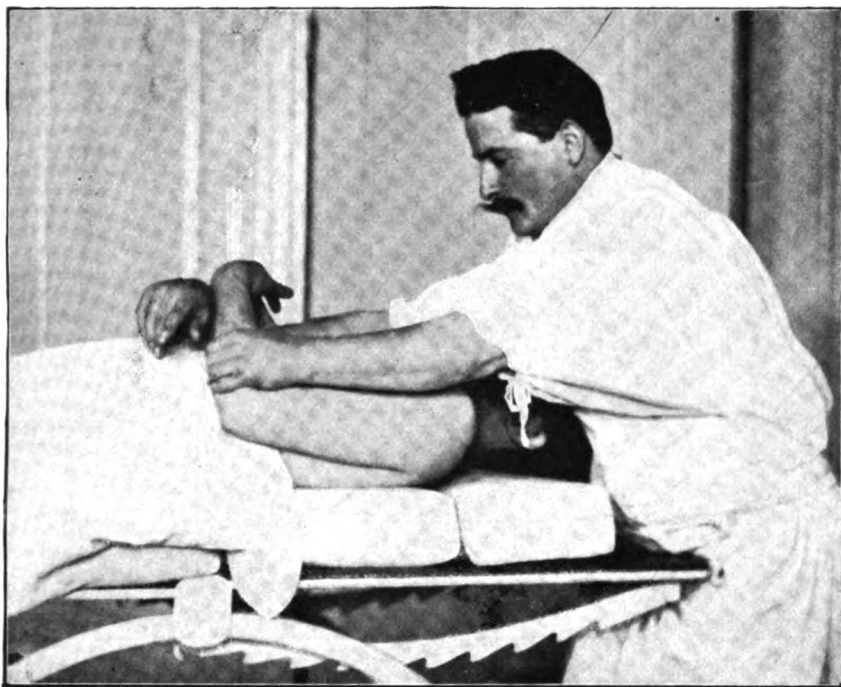


FIG. 1.—Method of performing artificial expiration. To produce expiration, both arms are pressed firmly to the thoracic walls.

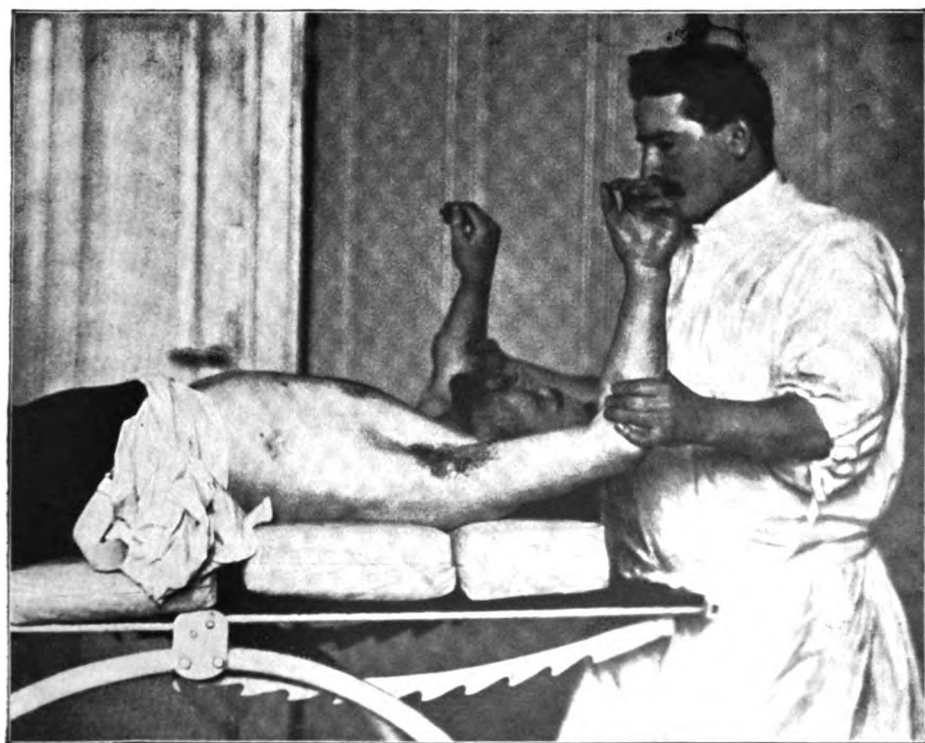


FIG. 2.—To produce inspiration, both arms are extended to their maximum extent.

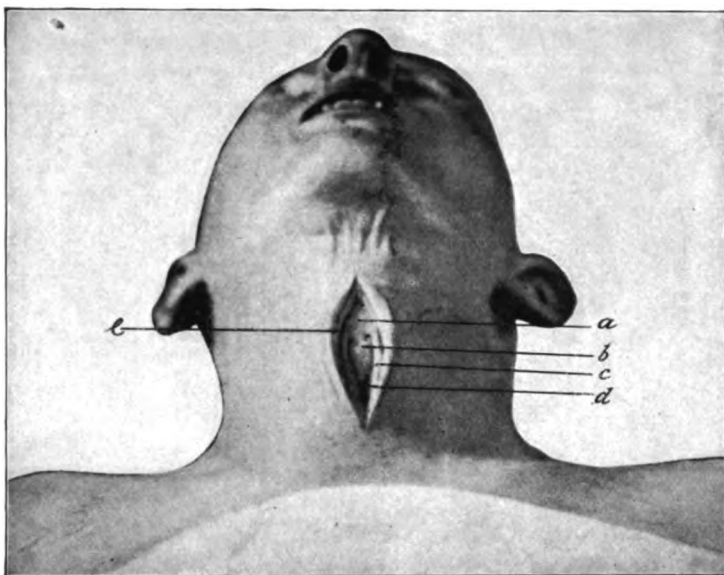


FIG. 3.—Superior tracheotomy (erico-tracheotomy): *a*, thyroid cartilage; *b*, cricoid muscle; *c*, cricoid cartilage; *d*, first tracheal ring; *e*, retracted thyroid.

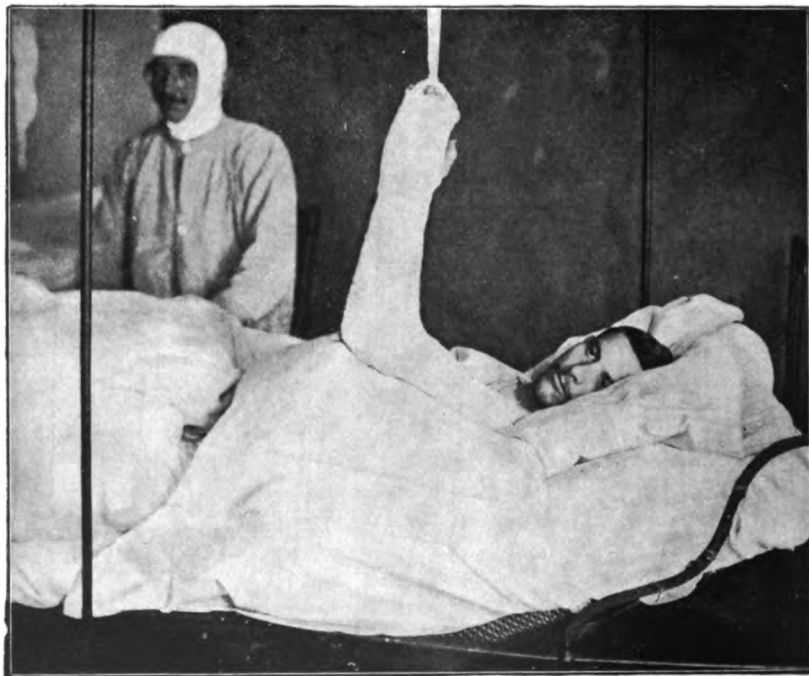


FIG. 4.—Volkman's method of suspending arm while in a splint resting on a pillow.

of the tongue, and injections of salt solutions have given good results.

The question of how long these procedures shall be kept up, naturally depends upon the case itself. They certainly should not cease until all hope of deriving any benefit therefrom has ended, which should never be before a half-hour, unless respiration is re-established. Those cases in which breathing stops but the pulse continues have a much more favorable prognosis than when the pulsations cease first.

Tracheotomy (Fig. 3.) may have to be performed in those patients in which the posterior crico-arytenoid muscle is paralyzed or the trachea stopped up by means of mucus, blood, or the contents of the stomach.

VOLKMANN'S SUSPENSION APPARATUS.

The patient I shall show you to-day is being treated for a septic phlegmon of the arm and forearm. It is a mistake to think that an arm, because it is infected, should not be treated with careful asepsis. All possible burrowing-places for pus must be drained and thoroughly washed with an antiseptic solution. The swollen skin is then lightly incised in numerous places, which lessens the tension. All of these spots should be carefully drained with wedges of gauze so as to produce capillary drainage. The wet dressing is applied, which is covered with gutta-percha paper so that evaporation may not take place. The outside bandage is to be loosely applied and the whole treated in Volkmann's suspension apparatus, which consists in a movable crane, the splint in which the arm is placed being held erect by a bandage attached thereto. It is surprising how much benefit is derived from a phlegmon treated in this manner. (Fig. 4.)

LIGATURE OF THE COMMON CAROTID ARTERY.

I have the opportunity to-day to show you some operations upon the cadaver. The ligation of the common carotid is made by incising the inner border of the sternocleidomastoid muscle at the height of the thyroid cartilage, on a line extending from the sternoclavicular articulation to a point midway between the mastoid process of the temporal bone and the angle of the jaw. After division of the skin, the platysma, and the fascia, the sternocleidomastoid

muscle is easily recognized by the direction of its fibres, and with a retractor are drawn latterly. The incision parallel to the muscle is now made deeper, until one recognizes in the under corner of the wound the omohyoid crossing the sternocleidomastoid. Immediately above this crossing point can be felt at the bottom of the wound the anterior tubercle of the transverse process of the sixth cervical vertebra (carotid tubercle), and upon this rests the common carotid artery, the internal jugular vein, and the pneumogastric nerve which lie in a common sheath. Upon the latter lies a small nerve trunk, the descending branch of the hypoglossal nerve. In opening the sheath, care must be taken not to injure the internal jugular vein, which in the majority of cases lies externally more or less close to the artery. This vein is carefully pushed to one side and the carotid elevated, care being taken not to include in the ligature the pneumogastric nerve. (Fig. 5.)

LIGATURE OF THE POSTERIOR TIBIAL ARTERY.

One has the choice of tying the posterior tibial artery in any portion of the lower two-thirds of the leg. If it is decided to perform the operation in the middle third, a longitudinal incision, eight to ten centimetres in length, is made about two centimetres in front of the internal border of the tibia, taking care not to injure the saphenous vein and the internal saphenous nerve. Both of these vessels are drawn upward and the medial border of the inner head of the gastrocnemius is at once recognizable. In front of and beneath the same lies the soleus, which joins with its fascia the tibial border. This must be separated, taking care to go in between the fleshy fibres of the soleus until the aponeurosis is reached. This membrane is then incised and the artery is seen to rest upon the posterior tibial muscle and on its lateral side accompanied by the posterior tibial nerve. The ligature of the posterior tibial artery in the lower third of the leg is made by a bow-shaped incision exactly in the middle between the internal malleolus and the tuberosity of the os calcis. Beneath the skin is found the lancinate ligament which strengthens the fascia suræ. As soon as this is separated, in exactly the same direction as the initial incision of the skin, the posterior tibial artery is found, usually accompanied by two veins, one of which is situated anteriorly, and the other posteriorly. If the incision is made too far posteriorly, the nerve is found and the

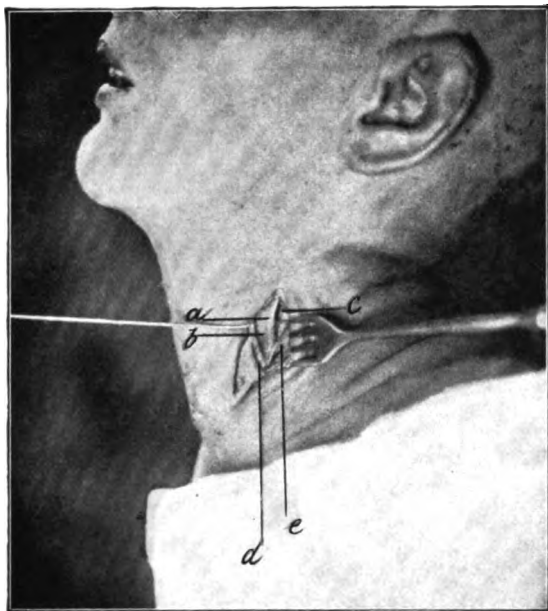


FIG. 5.—Ligature of the common carotid : *a*, carotid artery ; *b*, sixth cervical rib ; *c*, jugular vein ; *d*, omohyoid muscle ; *e*, sternocleidomastoid muscle.

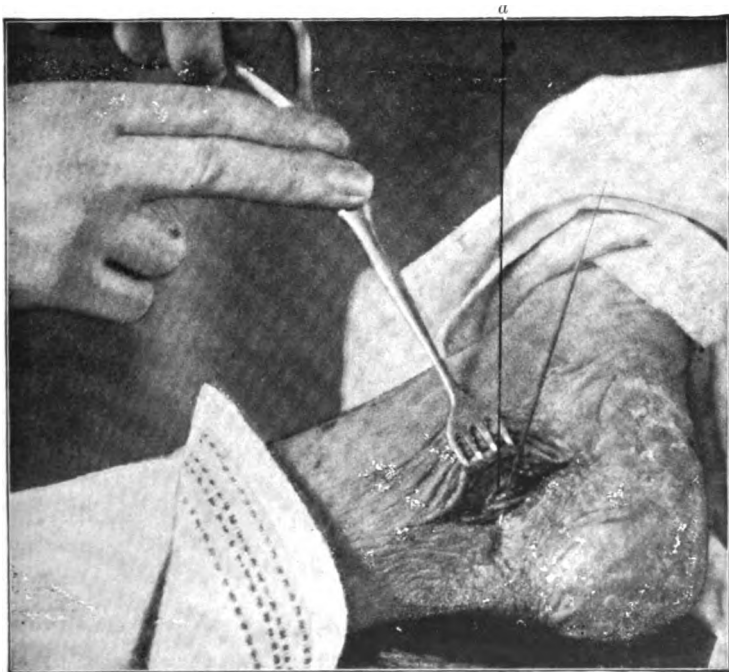


FIG. 6.—Ligature of the posterior tibial artery : *a*, border of the lancinate ligament. A sound has been placed under the artery and its accompanying vein.

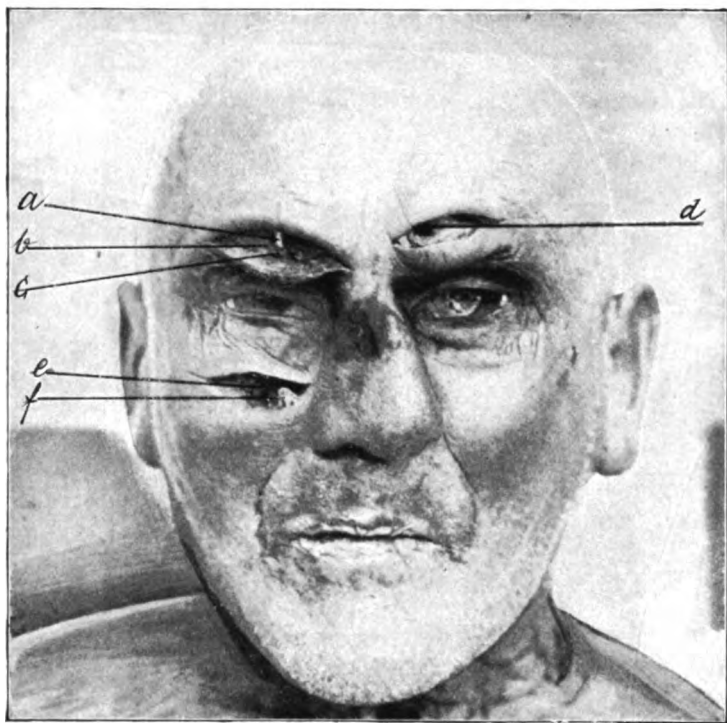


FIG. 7.—Opening of the frontal sinus. Resection of the supra-orbital and infra-orbital nerves: *a*, supra-orbital nerve; *b*, orbicularis muscle; *c*, supra-orbital artery; *d*, opening in the frontal sinus; *e*, orbicularis muscle; *f*, infra-orbital nerve.

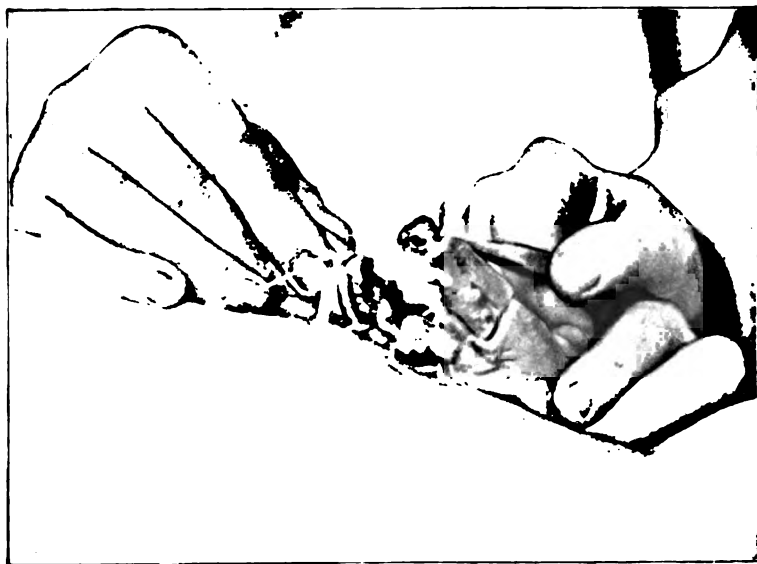


FIG. 8.—Resection of the bowel.

incision must be made somewhat more anteriorly. If the incision is made too far anteriorly, the tendon of the flexor longus digitorum is discovered and the artery must be searched for more posteriorly. (Fig. 6.) Whichever route is chosen, the ligature of the artery is performed in the usual manner.

OPENING OF THE FRONTAL SINUS.

The frontal sinus must sometimes be opened for collections of pus which have no means of being otherwise drained. Careful disinfection of the part is first to be practised, and any hairs close to the field of operation shaved off. The incision is made from the middle of the eyebrow and just above it, and continued to the median line, at once going down to the bone. In making this incision, the frontal and supraorbital nerves and vessels are incised, though by careful dissection this can sometimes be avoided. After displacement of the periosteum, the anterior wall of the sinus is opened with a small trephine. Considerable hemorrhage will take place because the anterior wall is partly composed of diploe. After separation of the anterior osseous wall, the bluish-red mucous membrane appears bladder-like in the opening. This membrane is usually thickened in collections of pus. The mucous membrane is now opened, and there may escape a large amount of pus. The diseased membrane is then scraped with a sharp spoon, and the sound is passed forward and backward in the natural opening of the sinus into the nasal cavity which is situated at the anterior end of the middle turbinated bone of the nose.

To secure permanent healing it is necessary to permit free drainage. For this purpose the normal opening is dilated, as described above, and a drainage tube inserted. When the opening is not found, a new passage-way must be made with the greatest care between the frontal sinus and nasal cavity with a trocar. Such an opening, however, remains patulous with more difficulty than the dilated normal opening. The whole sinus is then dusted with iodoform and packed with iodoform gauze. The external wound is sewed, with the exception of directly over the opening from which the end of the iodoform gauze may be left protruding, so that it may be removed in the course of three or four days, or repacked, as necessity requires. (Fig. 7.)

RESECTION OF THE SUPERIOR AND INFERIOR ORBITAL NERVES.

To resect the superior orbital nerve, an incision is made directly through the eyebrow which has previously been shaved off and the parts cleansed. The incisura supra orbitalis is easily found by the sense of touch. The nerve lies somewhat deeper than the accompanying artery, both of which rest upon the periosteum. It is very difficult to resect the nerve without injuring the artery. The nerve is best grasped with a small Thiersch forceps, well drawn out, and cut off.

To resect the inferior orbital nerve, an incision is made beginning about one-half centimetre beneath the end of the infra-orbital border and extending to the under border of the malar bone. The incision goes down to the bone separating the insertion of the quadratus labii superioris muscle. Above is seen the orbicularis oculi muscle. The periosteum is removed to the exit of the nerve from the infra-orbital canal. The infra-orbital artery is next separated by means of a chisel. The bony wall is removed, and the nerve is exposed and resected. This operation requires considerable skill in its performance and should always be practised upon the cadaver previous to its being done upon the living subject. (Fig. 6.)

RESECTION OF THE INTESTINE.

In resection of the diseased intestine it is of the utmost importance for healing that the portion sewed should be healthy. Kocher lays emphasis upon the mesenteric attachment being sufficient adequately to supply blood to the part, and this fact should always be borne in mind before the introduction of the stitches. In the resection of the mesentery *angular* portions are not to be removed, but rather the portions of the mesentery to be removed are to be cut *parallel* to the intestine. If for one reason or another no more intestine can be resected, an artificial anus is to be made with the upper end of the bowel. This is especially the case in the resection of the ascending and descending colon. The circular intestinal suture is performed as follows: The intestinal coils are surrounded with sterile towels so that the abdominal cavity is entirely shut off. Then, where resection is to take place, two clamps are attached almost perpendicularly to the long axis of the intestine, so that on the convex side of the bowel slightly more space is

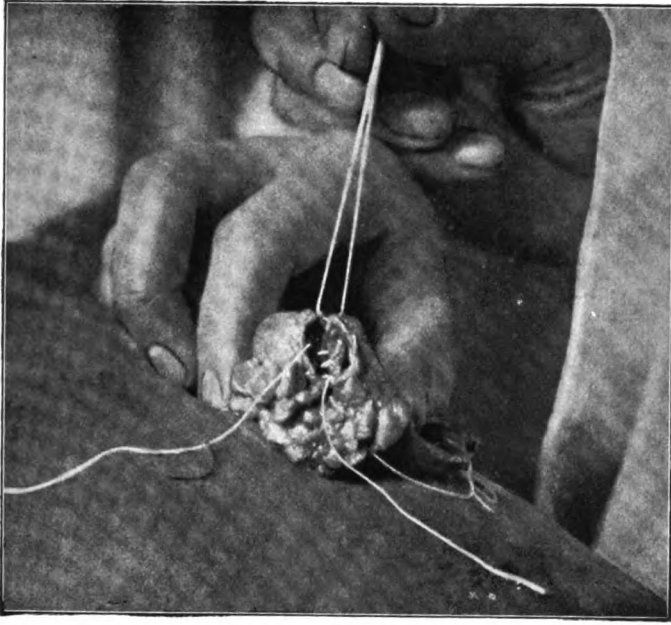


FIG. 9.—Resection of the bowel.

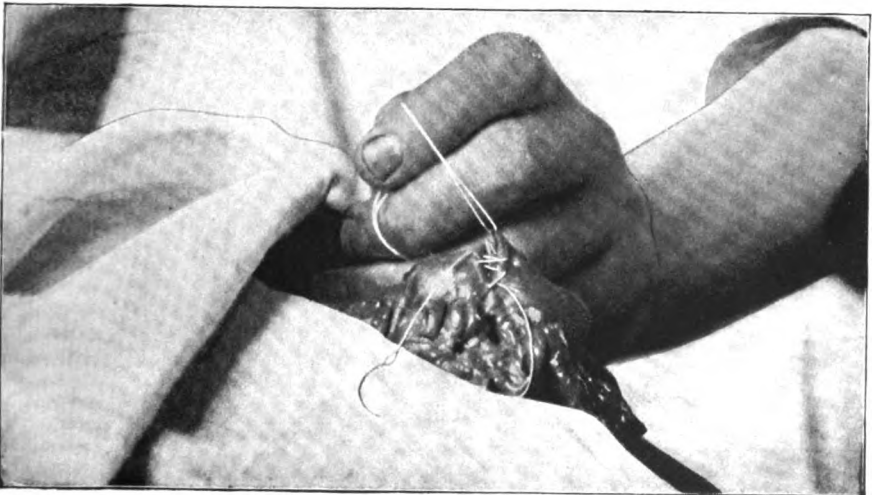


FIG. 10.—Resection of the bowel.

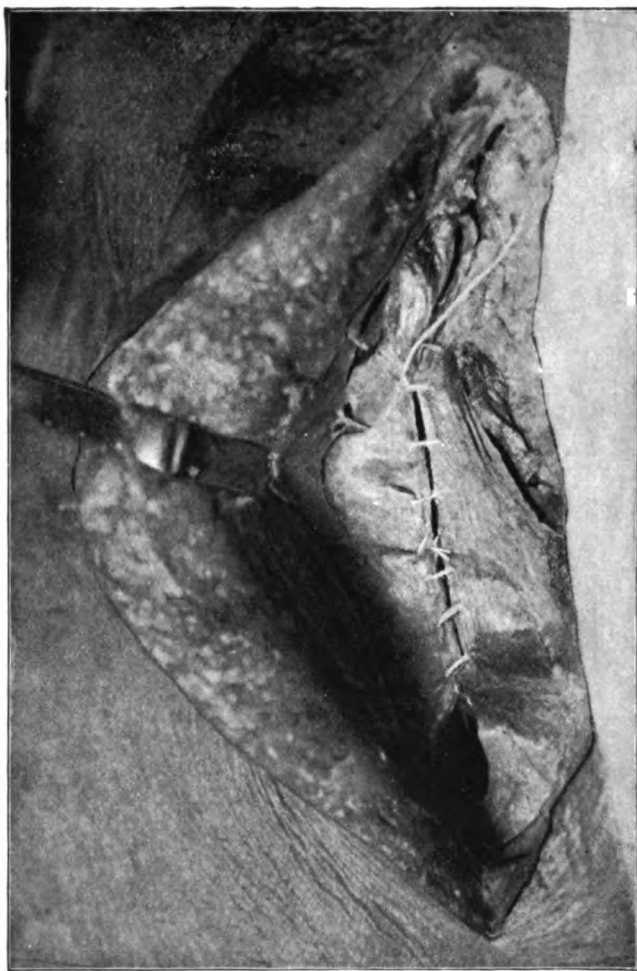


FIG. 11.—Sutures used in Bassini's radical operation for a left-sided inguinal hernia.

allowed than on the side attached to the mesentery. If a trained assistant is at hand, it is much better, however, that the lumina of the bowel above and below the portion to be removed should be shut off with his fingers. (Fig. 8.) Between these closed ends the intestine is separated, the cut surfaces washed with sterilized water, and the mesentery dissected close to its attachment to the intestine, with most careful attention to the removal of blood which may exude. The ends of the bowel are carefully sterilized, care having been taken to press back any fecal contents that might possibly exude. Sutures may be made individually or continuously. It is important, however, that two provisional stitches be made (Fig. 9) to mark the juxtaposition of the cut ends, one of which is placed at the mesenteric attachment, the other at the outer convex border. The stitches are made in such a manner that more of the serous surface is taken in than of the mucosa. The first sutures are made at the mesenteric attachment. Should the continuous sutures be used, the end of the thread introduced into the first stitch is left long enough to tie when the entire circular suture has been completed. The line of sewing is cleansed with warm sterilized salt solution, the sterilized towels are changed, and Lembert's suture applied (Fig. 10). For this purpose, fine silk and small needles are used, and the serosa and a portion of the muscularis alone are sutured, the stitches being about half a centimetre apart. Murphy's button is recommended in cases requiring despatch, and when the intestine is bound down by adhesions. The method of applying the continuous suture is seen in Fig. 11, where, after the application of the central stitch, the suture is started at the left and ends at the right.

Dermatology

RECURRING PHLYCTENULAR ERUPTION OF THE FINGERS, WITH CHANGES IN THE NAILS, POSSIBLY OF HYSTERICAL ORIGIN.

BY ARTHUR VAN HARLINGEN, M.D.,
Of Philadelphia.

Miss M. J., thirty years of age, consulted me July 23, 1883. She was thin and prematurely gray, of a neurotic temperament, suffered from constipation and "biliousness," and was subject to attacks of headache. She had suffered for six weeks past with a painful swelling around the nails of the left hand and the right thumb, occasionally attended by suppuration. Pain was the most prominent symptom, sometimes so severe as to keep her awake at night. Cauterization with nitrate of silver, soothing ointments, and lotions externally, with sulphur internally, had been employed, but without much effect.

On examination, the thumb, first, middle, and ring fingers were found to be affected. The tissues around the sides and root of the nails were swollen, red, and inflamed. Shooting neuralgic pains were felt in the affected fingers, particularly towards the tips, and the inflamed parts were exquisitely sensitive to the touch, but remained insensitive to the constant and the interrupted galvanic current. Applications of hot water, followed by lead water, were ordered; internally, a tonic aperient and one-sixth grain tablets of sulphide of calcium.

Three days later the note was made of little or no change in sensations. There was a slight purulent discharge around the edge of the nail-root. The following application was prescribed:

R Aluminis, gr. iii ;
Zinci sulphat.,
Plumbi acetat., aa gr. ii ;
Aqua, f ʒi.
Sig.—Apply locally.

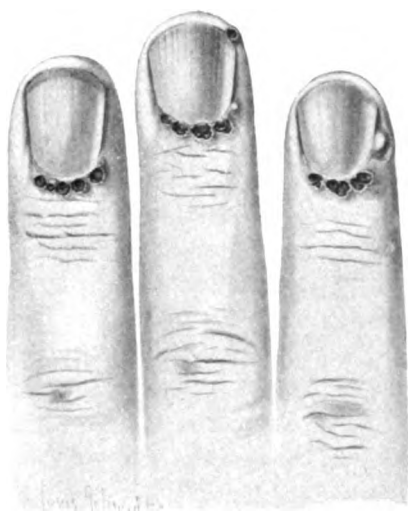


FIG. 1.—Recurring phlyctenular eruption of fingers. Appearance of fingers middle of August.

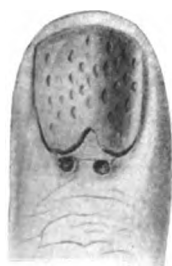


FIG. 2.—Appearance of thumb five months later.

This was followed by decided improvement in the symptoms. The skin around the nails was whiter and not quite so swollen. There was some pus along the edges of the nails, and around the nail of the ring-finger some pustules could be seen, but the skin was not so much everted at the edge as before. The sulphide of calcium was stopped, and quinine in tonic doses was prescribed. An ointment composed of liquor ferri subsulphatis, one-half fluidrachm, and unguentum aquæ rosæ, four drachms, was used on some fingers, but not much difference was noted between the effect of this and that of the astringent and sedative lotion.

On August 1 the patient was seen again, and was found to be suffering severe and continuous pain, unrelieved by anodynes. She appeared completely demoralized by the pain, and begged to have her nails removed by surgical operation. A combination of morphine, chloral, and bromide of potassium was ordered, and gave some relief to the incessant lancinating pains in the ends of the fingers, while a strong nitrate of lead ointment improved the appearance of the skin around the roots and sides of the nails.

On August 8 the patient's condition was worse. The inner side of the forefinger was so swollen and livid as to suggest the presence of pus. Incision was practised, but no pus was found, nor did the incision give any relief to the pain at first, although this gradually diminished; there were granulations about the root of the nail.

Two days later a similar livid swelling appeared on the side of the thumb, with one or two vesicopustules about the nail. The nails of the index- and ring-fingers began to become detached. Constipation was a marked feature, for which saline aperients with malt and quinine were given. The local treatment which seemed most effective at this period was the subsulphate of iron ointment, but often the patient could endure no application excepting that of lead water.

A few days later, the fingers were again observed to be swollen and puffy, with incipient granulations about the roots of some of the nails. Those of the ring-finger, middle finger, and index-finger began to separate, the separation beginning, apparently, by an effusion of pus under the middle, which gradually spread to each side of the root (Fig. 1). The nail then separated from the skin, and as it grew out was followed by exuberant granulations of the nail-bed,

which finally took the place of the cast-off nail. As this separation took place, the pain in the ends of the fingers diminished in intensity, although the patient's nervous and hysterical condition caused much hyperæsthesia and suffering.

While the nails of the thumb and first two fingers continued to separate from the nail-bed, new herpetiform vesicles and vesicopustules formed from time to time on the tips of the fingers. At one time the nail, which had been scraped thin, separated in the middle, or seemed to dissolve, showing exuberant granulations, which protruded from the orifice in its centre.

The patient went for a short stay in the country on August 27, but returned four days later, complaining of chills and feverishness, with pains in the back of the head and down the back. Her tongue was red and raw, and the mucous membrane of the buccal surface of the lips was covered with small blebs. There were a few new vesicopustules on the ends of the fingers, and even on the nail-bed where the nail had recently fallen off. Twelve grains of quinine were given. In a few hours the whole of the inside of the mouth, particularly the tonsils and fauces, was covered with small herpetiform vesicles, breaking down almost immediately into ulcers. There was a history of contagion, and this outbreak of herpetic angina had apparently nothing to do with the herpetiform paronychia. The face and nose also became the seat of an herpetiform eruption, but the acute affection ran a rapid course, and at the end of ten days had disappeared.

Shortly after, an abscess of the external meatus formed, which added to the patient's pain and discomfort. Calcium sulphide, one-tenth grain every two hours, was ordered, which seemed to give relief. The patient's general condition was wretched. She was emaciated, sleepless, and almost worn out. A milk diet, with the addition of beef-juice, was ordered.

On September 25 she was recorded as weak, forlorn, and nervous. There were hyperæsthetic areas in the region of the right clavicle and the left knee.

October 5.—Only granules of podophyllin are being taken. Milk diet continued. Her general condition was decidedly improved. New nails were now coming out in place of the diseased ones, which had not been shed, but slowly pushed off by the growth of the healthier nails. There was still considerable pain in the tips of the

fingers and around the nails. A few small pustules formed from time to time around the edges and root of the nails. The milk diet was continued, and daily salt baths were ordered. The aperient iron tonic was recommenced.

On October 11 continued improvement was noted. The new nails were nearly normal, but showed slight longitudinal ridges. The finger ends were red and showed occasional pustules. Spasmodic twitching of the muscles of the fingers was observed. A few days later the patient sent word she was suffering from "amaurosis," but this symptom was transitory.

The patient continued to improve, and soon after was sent for a short stay in the country, from which she returned in much better condition and in good spirits. The pains in the fingers were experienced only occasionally and to a slight degree, and the nails were almost entirely reproduced, but somewhat deformed and yellow towards the distal ends, with longitudinal grooves. Her hair began to fall rapidly at this time. There was a tendency to seborrhœic eczema of the nose and face.

There was a slight tendency to relapse on December 12, with recurrence of pain and the formation of a certain number of phlyctenulæ alongside the ring-finger nail, but this soon passed away and the improvement continued.

During January, 1884, the improvement in the nails continued, but they showed elongated, shallow, oval depressions (Fig. 2). Occasional but decreasing neuralgic pains were noted as long as the patient was under observation.

With the return to health the alopecia gradually ceased, and with the aid of stimulating applications to the scalp the hair had begun to grow rapidly by the middle of February. The color of the hair was prematurely gray when I first saw Miss J., but the new hair came out almost black in color at first; later it was of a dark chestnut color and quite abundant. The patient disappeared from observation March 24, 1884, and I have never heard of her since.

Reviewing this case, we have a woman of neurotic temperament, in whom severe attacks of pain referred to the tips of the fingers were accompanied by inflammatory local lesions, sometimes of a herpetic type, sometimes closely simulating an ordinary paronychia, and sometimes almost resembling a phlegmonous inflammation of

the ends of the fingers. The pain was of a paroxysmal character, lasting for hours or days with exacerbations, sometimes seeming about to cease and then suddenly almost without cause beginning again. It could not be accounted for by the inflammatory lesions, but occurred sometimes independently of them; and instead of being throbbing and local, it was of a darting, lancinating character, and extended at times up the forearm. The area involved was that supplied by the digital branches of the ulnar nerve, which suggested the probable involvement of that nerve, although there were none of the usual signs of a neuritis.

The patient was hysterical. Her symptoms were all exaggerated, and the pain and suffering of which she so bitterly complained did not seem to be accounted for by the external signs. I do not mean to say, of course, that her sufferings were not acute and real; there are few forms of disease where these are more so than in hysteria. But they did not seem justified by the local reaction, and the expressions of pain were always of that exaggerated character which we associate with the hysterical condition. Moreover, the peculiar resistance to electrical excitation, although this was not carefully tested, and the development at one period of hyperæsthetic areas, together with the curious attack of temporary blindness (the "amaurosis" reported), seemed to confirm the idea of an hysterical origin of the disease of the nails.

Investigation into the patient's general condition, and in particular the examination of the eyes and of the various alterations of sensation in the skin, was not carried out with completeness. It should be said in extenuation of these shortcomings, that the case was under observation nearly twenty years ago, when such examinations were not in general use, that the patient was not in a hospital, that no trained nurse was employed, and that her office visits were made at her own caprice. Moreover, my observation was naturally directed to the skin lesions, and at that time the connection between affections of the skin and hysteria had not attracted attention.

On looking over the case in retrospect, however, it has seemed to me that this peculiar phlegmonous and herpetic affection of the fingers, and of the finger-nails in particular, may well have been the result of hysterical influence. I have called attention to the various affections of the skin observed in the hysterical condition, in a paper read before the College of Physicians of Philadelphia in

1897.¹ In a later paper read at the meeting of the American Dermatological Association in the same year,² I reported four cases of neurotic excoriation of the skin due to the hysterical condition. The present case, it seems to me, may fairly claim a place in the category of hysterical dermatoneuroses. Other affections of the nails have been observed in connection with hysteria, although such cases are extremely rare.

Falconi³ reported a case where the patient, a markedly hysterical woman, suffered with pains along the ulnar nerve. Certain (unspecified) fingers became œdematous. The nails, particularly the thumb-nail of the right hand, became rough, scaly, and furrowed; the toe-nails also were affected. Blood and pus appeared under some of the nails, which dropped off, but without excessive pain at this time. The nails were regenerated later, but in a deformed condition. Delamare⁴ observed in two cases of hysterical women enlargement, thickening, and hardening of the nails.

Of the other similar affections of the nails and finger-tips, those found in erythromelalgia and in Raynaud's disease and its hysterical counterfeit may be mentioned. In some essential respects these cases differ greatly from that of Miss J., and need not at present be considered.

Mention may be made, however, of certain cases described chiefly by French writers, which have some points in common with mine. Hallopeau⁵ describes the case of a patient who had suffered from childhood with Raynaud's disease. For some months previous to the report, suppurative polydactylitis had been noted, with a generalized pustular dermatitis; the "pustulettes" formed were ephemeral, drying up within twenty-four hours. The local asphyxia had caused the polydactylitis, for it had spared certain fingers, and these were exempt from the suppurative lesions. The ephemeral outbreak of pustules over the general surface was supposed by Hallopeau to be due to resorption of either the pyogenic

¹ The Hysterical Neuroses of the Skin, American Journal of the Medical Sciences, July, 1897.

² Report of five cases of Erythematous Hysterical Dermatoneurosis, International Medical Magazine, November, 1897, p. 695.

³ Deutsche medicinische Wochenschrift, 1886, p. 717.

⁴ Nouv. Iconogr. de la Salpêtrière, November, 1896, p. 370.

⁵ Ann. de Derm. et de Syph., 1890, p. 420.

microbes or their toxins, and not to local infection. Their symmetry, and the fact that each outbreak coincided with the suppuration about the nails, seemed to prove this. The polydactylitis was followed by marked alterations in the nails.

Stowers¹ reports the case of a woman who suffered after child-birth with periungual abscess of the thumb and fingers, developing gradually and successively during a series of years. A pustule formed in the internal border of the nail-bed, and consecutive ulceration destroyed the matrix. Itching, burning, and lancinating pains were experienced. The toes also were affected, and the disease lasted for forty-five years, until the patient's death from another cause.

Audrey² describes a number of cases of somewhat similar character, but differing in many important respects from that which I have reported. One or two, however, closely resemble it. A woman of forty-nine was seized with violent pain, accompanying the appearance of a phlyctenula filled with pus on the edge of the right thumb-nail. At the end of some weeks the nail was shed, and the pain became much less severe. Several months later the same kind of pains with suppuration involved the right middle finger, followed by shedding of the nail. Then followed the right index-finger. The fourth nail to be attacked was the left index; then came the turn of the left thumb, and successively all the other fingers except the little finger of the right hand, which remained healthy. During all this period the patient suffered insupportable pains, involving the internal aspect of the upper arms, from the axillæ downward. The pains lasted five or six days and then ceased. When examined by Audrey, twelve years after the commencement of the disease, there had been no lymphangitis, no chilblains, and no trace of local asphyxia or of stasis. The hands were white and normal, without atrophy or anæsthesia. The nails had disappeared; the fingers were pointed, and rough over the nail-bed, with occasional pustules. The right little finger alone remained intact. The right great toe-nail had dropped off, but otherwise the feet were normal.

Audrey reports a number of other cases, but none so closely

¹ *British Journal of Dermatology*, 1896, p. 1.

² *Les Phlyctenoses récédévants des extrémités*, *Annales de Derm. et de Syph.*, November, 1901, p. 913.

resembling mine. He divides the cases into groups, some of which are very different in appearance, course, and symptoms from those just quoted. From an analysis of the group to which belong those cases most nearly resembling that of Miss J., Audrey draws the following conclusions: (1) The disease begins constantly and often spontaneously on the extremities of the fingers. (2) Little lakes of pus form, more or less extensive, roundish, unelevated, lodged in the thickness of the epidermis, covered with desquamation, and leaving after them transitory erosions, not ulcers; in other words, purulent phlyctenulæ, not pustules. (3) The tissues are affected almost as in ordinary panaris or paronychia. (4) The disease extends to the palm, then to the back of the hand; the same is true of the feet. (This was not the case with Miss J., nor in the cases I have quoted from Audrey. It differentiates these from his type.) (5) Limitation of the disease to the hands and feet; the latter less severely affected, and not necessarily involved. (6) Moderate local pruritus, local pain, sometimes intense; in exceptional cases, violent pain irradiated on the internal surface of the arm. (7) The slow progress of the disease; its persistence, amounting to incurability. (8) The absence of general disturbance of the health. (9) The absence of septic, lymphatic, or other complication, atrophy, anæsthesia, etc. (10) Finally, the transformation of the digital extremities, shrivelled, deprived of their nails, and reduced to small, conical, sclerosed stumps. (Miss J.'s case was not long enough under observation to indicate whether or not this result would take place.)

Audrey says that although we know nothing positively of the pathogenesis of this affection, or rather of these phenomena, everything seems to point to an original neuritis. The extraordinary course of the disease, the violent neuralgic pains, and particularly the positive lesions encountered in these cases, all point in this direction. Infection plays a constant but secondary part.

The same view is taken by Carle,¹ who, remarking upon a case he reports, says that the affection is probably a neuritis, rather than a suppurative polydactylitis of septic origin or a trophoneurosis of central origin. The lesions, though varying greatly in different cases, occur generally in the track of certain terminal nerve-fibres.

¹ *Annales de Derm et de Syph.*, February, 1902, p. 130.

Ophthalmology

THE CLINICAL SIGNIFICANCE OF BINOCULAR DIPLOPIA.

BY WILLIAM T. SHOEMAKER, M.D.,

Assistant Ophthalmologist and a Chief of Clinics to the German Hospital;
Dispensary Ophthalmic Surgeon to the Presbyterian Hospital,
Philadelphia.

THE fundamental principle of perfect binocular vision is that each eye shall receive an image of the object looked at upon points of the retinae which are *corresponding* or *identical*. Every point in the visual retina of one eye has such a point correlated or correspondent in the retina of the fellow-eye. Similar images of the object at these points are fused or united by the cerebral centres, and the object is seen as one. If, however, the images fall upon retinal points not identical, fusion does not take place; each image is referred to cerebral centres for that eye and separately recognized,—diplopia results. Granting that the two eyes have previously enjoyed binocular fixation,—*i.e.*, an object looked at has produced on each eye an image at the fovea centralis, or yellow spot,—the appearance of diplopia means that, whereas the image in one eye is formed at the fovea (the fixing eye), that in the other eye is not so situated, and is therefore not at an identical point.

Retinal points in the two eyes previously identical have become *disparate*. Such a disturbance may be brought about by mechanical causes which are readily divisible into two general classes.

Causes of the first class tend to force the eyeball into a *new position as a whole*; the eyeball is translated, and is no longer in a position harmoniously to receive images with its fellow-eye.

Causes of the second class are those affecting the musculature of the eyeball. The eyeball, while retaining a position in the orbit which in itself may not be abnormal, suffers some muscle defect

whereby it is deviated, and is incapable of following the movements of the other eye throughout the full field of fixation.

The relegating of diplopia to the one or the other of these two general classes of cause is an important diagnostic step, for the individual causes active under each class may be, and generally are, totally different, and call for treatment just as different.

Diplopia, it must be remembered, is only a symptom mechanically produced, and one, too, not pathognomonic of any one disease. It is, however, a very valuable and important symptom, and when suddenly appearing in an individual, is subjectively so annoying that it is almost certain to bring that individual at once under medical examination.

It is the object of this paper to deal with the principles involved, attention to which it is hoped will enable the examiner to handle with intelligence such cases presenting, and not be content to rest satisfied with the patient's statement that he sees double.

The first step, then, is to place the diplopia within one or the other of the above-mentioned classes. It becomes necessary to eliminate or confirm a new position of the eyeball. A critical examination of the face and of the eyes in the primary position—*i.e.*, looking directly forward to some point far enough removed to be regarded as at infinity (not less than five or six metres)—will show in many individuals asymmetry. Asymmetry may be so marked as to be misleading, if carelessly observed, in certain cases in which we are endeavoring to determine if one or the other eyeball occupies a false position. One side of the face may be well developed, while the other is undeveloped, smaller, and less prominent. Such cases do not, on account of the asymmetry, have diplopia, although the eyeballs may occupy quite different planes. They have developed that way, and as they developed, retinal points likewise developed identically. If the two sides of the face are separately examined, whereas they will be found different, each in itself may be normal. Observe, then, the following points: Are the superior and the inferior palpebral folds, the palpebral fissure, the movements of the lids, and the positions of the lachrymal puncta well proportioned and normal to that side of the face? If they are, it is fair to presume that the eyeball occupies the position in the orbit which it has always occupied, for the tendency of a new position is to alter these relationships. Any false position of the eyeball may be met with,

and attention to the points above mentioned will generally suffice to confirm the diagnosis in those cases which are not sufficiently pronounced as to speak for themselves. Forward displacement is known as exophthalmos, or proptosis. Exophthalmos may be directly forward, or other directions, such as upward, downward, inward, or outward, may be added thereto. The tendency in exophthalmos is towards obliteration of the palpebral folds and widening of the palpebral fissure. If the eyeball is sufficiently proptosed, complete closure of the lids becomes impossible. Lachrymation and perhaps epiphora are apt to result from the disturbed relationship between the lachrymal puncta and the eyeball. The former fail accurately to apply themselves against the latter, rendering inefficient the capillary action by which the tears are normally led into the lachrymonasal canal. Backward displacement of the eyeball is known as enophthalmos. The eyeball recedes into the orbit either directly backward or backward and downward. In this condition the superior and the inferior palpebral folds are accentuated, and the palpebral fissure is narrowed. The lachrymal disturbance is the same, and for similar reasons as in other malpositions of the eyeball. Excursion of movement may or may not be limited in both exophthalmos and enophthalmos.

In all cases in which an eyeball has recently assumed a new position in the orbit, diplopia is to be expected, provided that each eye has a sufficient amount of vision, that the line of vision for each eye is not obstructed, as by ptosis, paralytic or mechanical (œdema of the lids, closure from swelling), and that the patient has not compensated for the defect by certain positions of the head, whereby identical points in each retina are brought into harmony. Such compensation is instinctively sought, and if possible readily found. The foregoing are the principal points of value in establishing the diagnosis of a new position of the eyeball.

The possible causes are many and need only be mentioned here, discussion of them being beyond the scope of this paper.

Traumatism has few limitations in its results. The eyeball may be forced primarily or secondarily into any position. The orbital walls may be fractured and pieces of bone driven inward, diminishing the orbital capacity or even directly pushing aside the eyeball. Likewise fracture may increase the size of the orbit, and together with other changes give rise to enophthalmos. Intra-

orbital hemorrhage, intra-orbital abscess, and foreign bodies within the orbit are causes of exophthalmos following injury. Thrombosis of the cavernous sinus, or of the orbital veins, orbital aneurism, tumor, and Graves's disease, which it should be remembered may occur with unilateral exophthalmos, must also be considered when an eyeball is found proptosed.

We now come to the consideration of diplopias of the second class; those due to faulty muscle equilibrium. It is in these cases that we must rely upon a careful study of the double images for a positive diagnosis of the individual muscle or muscles involved. Before this can be done, a thorough knowledge of the musculature of the eye and of the laws of visual projection is essential.

Each eyeball has attached to it six muscles, four of which are more or less direct in their actions and are called the straight or recti muscles; the remaining two, the oblique muscles, rotate the eyeball around an anteroposterior axis; they have also other actions. The exact actions of these muscles are shown in the following table from Duane.¹

Muscle.	Field of action limited to	Moves eye laterally.	Rotates upper end of vertical meridian (torsive effect).	Lateral and torsional effects increasing as eye is	Lateral and torsional effects diminishing to zero as eye is	Moves eye vertically.	Vertical action increasing as eye is	Vertical action diminishing as eye is
External Rectus.	Outer half of field of fixation.	Out.	No action.	No action.
Internal Rectus.	Inner half of field of fixation.	In.	No action.	No action.
Superior Rectus.	Upper half of field of fixation.	In.	In.	Adducted.	Abducted.	Up.	Abducted.	Adducted.
Inferior Rectus.	Lower half of field of fixation.	In.	Out.	Adducted.	Abducted.	Down.	Abducted.	Adducted.
Superior Oblique.	Lower half of field of fixation.	Out.	In.	Abducted.	Adducted.	Down.	Adducted.	Abducted.
Inferior Oblique.	Upper half of field of fixation.	Out.	Out.	Abducted.	Adducted.	Up.	Adducted.	Abducted.

¹ "Motor Anomalies of the Eye," p. 6.

For clinical purposes, the classification of Duane seems to be practical and readily adapted to the study of cases. He divides the twelve muscles of the two eyes into three groups of four muscles each. Four move the eyes laterally, four move them upward (elevators), and four move them downward (depressors). Each group is subdivided into two pairs, one muscle of each pair being in the right eye and the other in the left eye. The muscles of each pair are known as associated antagonists; each moves the eye to which it belongs in exactly the same direction and to the same extent as does its fellow move the eye to which it belongs.¹

The twelve muscles may be arranged in tabular form as follows:

<i>Acting laterally.</i>	{	External rectus, O. D.	}	Associated antagonists.—Right turners.
		Internal rectus, O. S.		
	{	External rectus, O. S.	}	Associated antagonists. — Left turners.
		Internal rectus, O. D.		
<i>Elevators.</i>	{	Superior rectus, O. D.	}	Associated antagonists.—Right elevators.
		Inferior oblique, O. S.		
	{	Superior rectus, O. S.	}	Associated antagonists. — Left elevators.
		Inferior oblique, O. D.		
<i>Depressors.</i>	{	Inferior rectus, O. D.	}	Associated antagonists.—Right depressors.
		Superior oblique, O. S.		
	{	Inferior rectus, O. S.	}	Associated antagonists. — Left depressors.
		Superior oblique, O. D.		

The elevators and depressors are divided into right and left, because in right and left elevation and depression the pairs so designated develop their greatest vertical action (Duane).

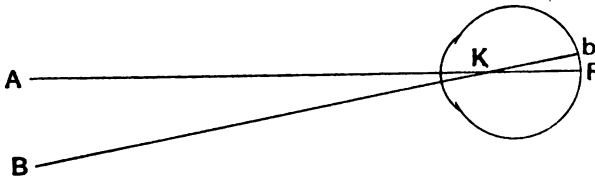
Now, to determine if a certain muscle is paralyzed, we must test the action of that muscle. This is done by so directing the eyes as to call into full play the suspected muscle. If the muscle is paralyzed, the eye will naturally fail to fulfil the tested function; it will, if the fellow-eye be sound, lag behind, and, of course, will lag the farther behind the farther in that direction the fixation point is carried.

The paralyzing of one or more muscles attached to the eyeball

¹ *Vide*, "The Diagnosis of Ocular Paralysis," by Alexander Duane, M.D., Ophthalmic Record, vol. x., No. 12.

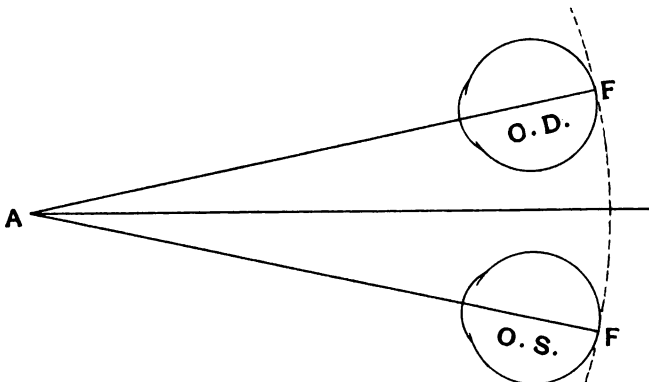
gives that eye over more or less to the antagonistic muscles, causing perhaps a squint or deviation, but however apparent this may be, we are still unable to detect the faulty muscle without, as above stated, attempting to carry the eye in such a direction as to call into play that muscle. Roughly, this can be done by having the patient, with his head erect and fixed, attempt to fix and follow the finger or any object held in front and moved successively in various directions. Any marked failure of one eye to follow in a certain direction can be seen, and the diagnosis placed within a certain group of muscles; but for a more accurate diagnosis we must have recourse to the study of the double images.

FIG. 1.



In Fig. 1 the eye is fixing the object A,—i.e., A forms its image on the retina at F, the fovea centralis. The ray A F is projected back over its course of entrance through K, the nodal point of the lens, and A is seen in its true position. The object B, situ-

FIG. 2.



ated to the left of A, sends its rays through the nodal point K, and casts its image at b. Projection takes place over the same course and B is seen in its proper place. It will be seen, however, that the

object B, situated to the *left* of A, has its retinal image to the right of the fovea which contains the image of A. The same reversal would, of course, hold true for objects situated in any direction from those in the visual axis A F.

Fig. 2 shows the two eyes fixing on A, the retinal image in each eye being at the fovea centralis, identical points; A is seen single.

FIG. 3.

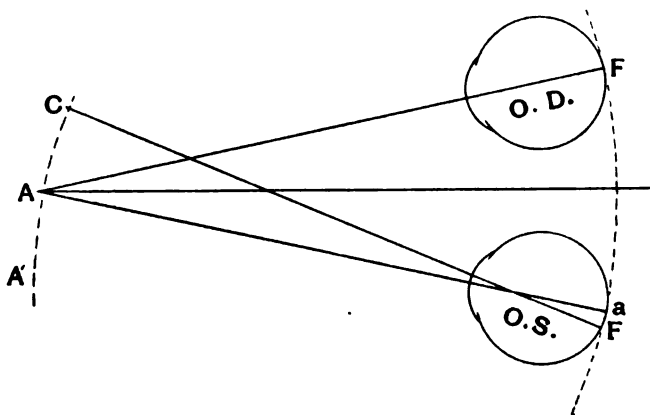


Fig. 3 shows the right eye (O. D.) fixing A, and the left eye (O. S.) turned in so that its visual axis takes the direction F C. A, therefore, forms its image on the retina of O. S. at a point (a) situated to the right of F, and now identical with F of O. D. Diplopia therefore results.

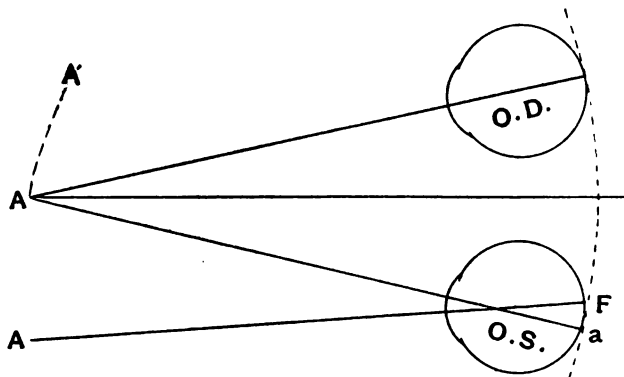
Now, O. S. having previously been in its normal position, with its fovea identical with the fovea of O. D., the higher perceptive centres consider it still so, and, knowing from experience that an image to the right of the fovea comes from a point to the left of fixation (Fig. 1), believe A to be situated at A', a manifestly false position. This image, then, is called the false image and belongs to the errant eye.

Fig. 4 shows O. D. fixing A, O. S. divergent. A forms its image in O. S. at a to the left of F, and for the same reason as above is projected falsely to A'.

These principles apply equally to any deviation of the eye. A glance at Figs. 3 and 4 will show at once that when the eyes are *convergent*, the right-hand image belongs to the right eye, and the left-hand image belongs to the left eye; when *divergent*, the right-

hand image belongs to the left eye, and the left-hand image belongs to the right eye. When image and eye correspond, the diplopia is *homonymous*; when not, it is *heteronymous* or *crossed*. Furthermore, diplopia within the field presided over by the lateral muscles

FIG. 4.



(adductors and abductors) is known as *horizontal diplopia*; diplopia within the fields of elevation and depression is known as *vertical*.

Knowing these principles and the muscles employed in carrying the eyes in any given direction, it is theoretically quite easy to locate the defective muscle. In cases where a single muscle is paralyzed, or several muscles in one eye are paralyzed, very little difficulty arises; but when muscles in both eyes are paralyzed the problem becomes more complicated, and in some cases almost impossible of perfect solution.

In the study of the double images a candle-flame is used; and to know always during the examination to which eye belongs each image, a colored glass, preferably red, is placed before one eye. Within the field of diplopia the patient then sees a yellow flame and a red flame. The true image is always more distinct than the false image; so by placing the red glass before the fixing or sound eye, when recognized from the start, a greater equality in the intensity of the two images is obtained. The head of the patient must be kept fixed in the primary position and not moved or turned to follow the candle to its different positions. The patient will instinctively move his head, and if this is not carefully guarded against, the examiner will be deceived as to the true condition. The object, it must be remembered, is to test the *movements* of the eyes. Start-

ing with the lighted candle in the median line directly in front, a note is made of the patient's answers as to the presence or absence of diplopia. If diplopia, the relation of the images to each other must be noted as to the following points: Which one is to the right and which one is to the left; if not on the same level, which one stands higher; whether they stand parallel; and if not, which one leans, and the direction of its inclination; and, finally, the distance between the two images. The candle is then carried far to the right, then to the left, and in order directly upward, directly downward, upward and to the right, upward and to the left, downward and to the right, downward and to the left. In each of these positions notes are made as above. Although not necessary, it is well to return the candle to the central position each time before carrying it to a new position.

With these data, a knowledge of the twelve muscles, and a knowledge of the laws of projection, a satisfactory diagnosis can in most cases be made. The practical points to be borne in mind are, that homonymous diplopia indicates abnormal convergence, crossed diplopia indicates abnormal divergence. In upward rotation the higher image belongs to the lower or lagging eye. In downward rotation the lower image belongs to the higher or lagging eye. If an image is leaning to the right (patient's right), the eye to which it belongs is abnormally rotated to the left, and *vice versa*. The distance between the two images increases as the candle is carried in the direction of the action of the defective muscle. Suppose, for example, we have paralysis of the external rectus muscle of the right eye. We would have horizontal diplopia manifesting itself so soon as any effort is made to carry the eyes to the right. It would be homonymous, because the right eye, not being able to turn to the right, would stand still, while the left eye, being sound, would turn to the right. The two visual axes would thus cross. Also the more the left eye turns to the right the greater would be the crossing or convergence; hence the greater would be the distance between the double images. As the internal and external recti muscles are direct in their action in the horizontal plane and do not in any way rotate the eyes, there would be no leaning. With these data, the affected muscle is at once located in the right eye and must be the external rectus of that eye. No other paralyzed muscle could give this combination. Diplopia became manifest when the eyes were turned



FIG. 5.—Compensatory position of the head when the field of diplopia is to the left. (Left abduction.)



FIG. 6.—Compensatory position of the head when the field of diplopia is up and to the left. (Left elevation.)



FIG. 7.—Compensatory position of the head when the field of diplopia is down and to the right. (Right depression.)

directly to the right. Now, the muscles active in this associated movement are the external rectus of the right eye and the internal rectus of the left eye. Suppose the paralysis affected the internal rectus of the left eye, this eye would then fail to follow, or would lag when looking to the right. The visual lines would, therefore, become divergent; the diplopia would be crossed, thus showing at once the lesion to be in the left eye and in the internal rectus muscle.

If an eye is incapable of moving in a certain direction, it is only natural that its possessor should avail himself of any compensatory power which he might have to obviate in the interest of single vision the necessity for such a movement. This he often does by turning his head in the direction of the action of the paralyzed muscle, or towards his field of diplopia. Thus, in the patient represented in Fig. 5, the left external rectus is paralyzed. His field of diplopia is to the left; he carries his head rotated strongly to the left. By doing this, both eyes, although fixing directly in front of him, are, relative to his head, deviated far to the right, rendering unnecessary any action on the part of the left turners. Figs. 6 and 7 show the compensatory positions of the head in vertical diplopia; Fig. 6 shows the position assumed when the field of diplopia is up and to the left; Fig. 7 shows that assumed when the field of diplopia is down and to the right.

Valuable hints can often be gotten by observing the position in which the head is carried, and in the presence of any striking deviation a careful search should be made into the muscle equilibrium with the head in the normal position. Diplopia may be present for a very brief period before the patient learns to correct it, and having once learned, if not a careful observer, he may consider himself cured, as it were, and even forget that he ever saw double.

Biographical Sketches

OF

EMINENT LIVING PHYSICIANS.

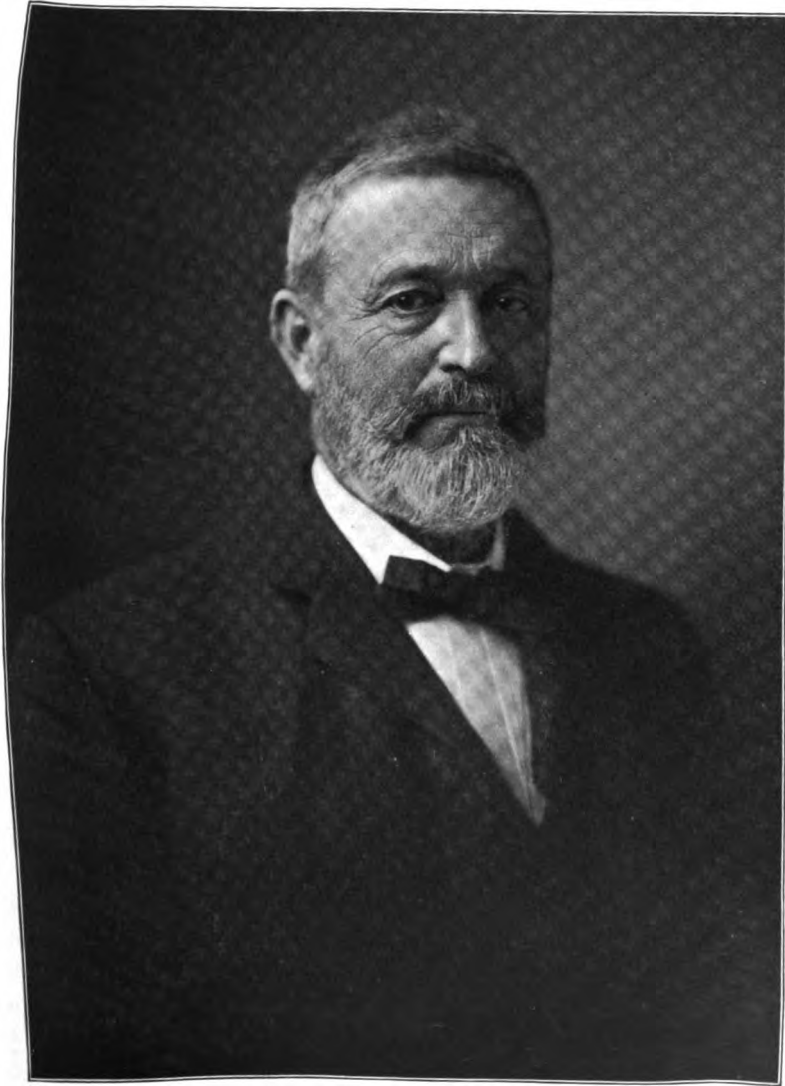
BY GUY HINSDALE, A.M., M.D.,
Of Philadelphia.

HORATIO C. WOOD, M.D., LL.D.

THE name of Wood has long been famous in American medicine. During the middle portion of the last century Professor George B. Wood occupied a conspicuous place as a teacher, a practitioner, and an author, and his name is identified with one of the most admirable treatises on medicine that has ever been published. It was under such tutelage that his nephew, the younger Wood, pursued his studies and equipped himself for the distinguished position which he has attained.

Graduating from the Medical Department of the University of Pennsylvania in 1862, Dr. H. C. Wood served as a resident physician in the Philadelphia Hospital and immediately after in the Pennsylvania Hospital, from which institution he went into the United States service in the hospitals in Washington and subsequently in Virginia. Before graduation he had served as assistant resident in the Friends Asylum for the Insane, so the foundation of his future life was laid in an extended and varied hospital experience.

In the early part of his life Dr. Wood gave much time to the study of natural history, devoting his attention especially to entomology and to botany; publishing in the "Smithsonian Contributions to Knowledge" and various other scientific publications a number of papers, the most important of which was his monograph of the "Fresh-Water Algae of North America," with nineteen colored and two uncolored plates, made from three hundred and sixty original microscopic drawings.



Horatio C. Wood, M.D., LL.D.
(From a recent photograph taken especially for the CLINICS.)

His work was soon, however, directed towards pharmacology, as was shown in a series of papers which attracted so much attention that by the time he was thirty-two years of age professorships in the Bellevue Hospital Medical College and the College of Physicians and Surgeons in New York had been offered to him, which were declined on account of his decision to remain in connection with the University of Pennsylvania, in which institution he had been made professor of medical botany in 1866.

While a resident in the Pennsylvania Hospital Dr. Wood's attention had been attracted by the numerous cases of sunstroke coming into the wards, and conceiving that the anatomical changes which had been reported from time to time as characteristic of the disease were in large part the outcome of post-mortem changes caused by high temperature at the time of death, he made examinations within two or three hours after death and demonstrated the correctness of his theory. His continuing interest in the subject led in 1872 to the "Boylston Prize Essay on Thermic Fever, or Sunstroke," in which the natural history, pathology, and rational treatment of the disease were first worked out so thoroughly that nothing radical has been added to the knowledge of the subject since. Subsequently, extending the scope of his research, Dr. Wood published in the "Smithsonian Contributions to Knowledge" in 1880 a research occupying two hundred and fifty-eight quarto pages on fever, a paper to which, perhaps, was chiefly due his election to the National Academy of Science.

In 1874 Dr. H. C. Wood published a treatise on therapeutics, which was immediately and continuously successful, reaching its eleventh edition in 1900. In this treatise he challenged the entire system of empiricism in medicine to which our profession had clung for two thousand years. He was firmly convinced that therapeutics developed in this manner cannot rest upon a secure foundation. He denies that clinical experience is the mother of wisdom, saying: "She has been in medicine rather a blind leader of the blind; and the history of medical progress is a history of men groping in the darkness, finding seeming gems of truth one after another, only in a few minutes to cast each back to the vast heap of forgotten baubles that in their day had also been mistaken for verities. In the past there is scarcely a conceivable absurdity that men have not tested by experience and for a time found to be the thing desired. . . .

Looking at the revolutions and contradictions of the past, listening to the therapeutic Babel of the present, is it a wonder that men should take refuge in nihilism, and, like the lotus-eaters, dream that all alike is folly,—that rest and quiet and calm are the only human fruition?"

Although man and the lower animals may present differences of susceptibility, Wood recognized that there is no difference in the mode of impression when drugs are administered to man and animals alike, thus establishing the value of the experimental test. On such a fundamental principle hangs all the law of modern pharmacology. It established and justifies the use of animals in the laboratory, without which little can be accomplished in developing our science and removing it from the darkness in which our predecessors groped. Wood's treatise was, we believe, the first book on pharmacology in any language to be avowedly based upon physiological investigation.

As long ago as 1833 Dr. George B. Wood published, in connection with Dr. Franklin Bache, the first edition of the "United States Dispensatory," becoming sole editor of the book after the death of his co-worker. When the time came for the preparation of the fourteenth edition Dr. George B. Wood had reached the age of eighty, and very naturally, when about half-way through the work of the edition, gave out, finally consenting to transfer his labors to his nephew, who, unaided, completed the task. When the fifteenth edition appeared, Dr. H. C. Wood associated with himself Professor Joseph P. Remington, the noted pharmacist, and under their co-working the "Dispensatory" was recast and so largely rewritten as to be practically a new book, whose success was immediate. Four editions of the book have appeared. The superiority of American pharmacy over that of the rest of the world has at least one of its roots in this pharmacological classic.

In 1870 Dr. Wood was appointed visiting physician in the Philadelphia Hospital, became lecturer on nervous diseases in the University of Pennsylvania, and was made a clinical professor of nervous diseases in the University in 1875. In 1876 he was made professor of materia medica and therapeutics in the same institution, resigning at that time his chair of botany.

Dr. Wood published in 1880 a work on "Nervous Syphilis," and in 1887 a work on "Nervous Diseases and their Diagnosis."

In 1896 he published, in connection with Professor R. H. Fitz, of Boston, a treatise on the practice of medicine.

Dr. Wood's influence in the development of the University of Pennsylvania has been felt in many ways. He was very largely instrumental in bringing about the modern form of medical education adopted twenty-five years ago and elaborated so successfully in later years. Dr. Wood's paper on the Medical Department of the University of Pennsylvania produced a storm which cleared the atmosphere; a new course was taken by the university, and other institutions soon followed in its wake. Dr. Wood was the first to suggest the idea of a University Hospital in connection with its school of medicine, and he secured the first large subscription, which gave the necessary encouragement in building and equipping that indispensable adjunct to the Medical Department.

His connection with the Philadelphia Hospital as an attending physician gave him unusual facilities for clinical teaching, and his lectures there were always highly appreciated by the students. But it is interesting to note that his outspoken criticism of the outrageous political management of that institution under the old régime three times cost him his position, but three times was he restored as political changes ensued, and that without the slightest overtures on his part.

Dr. Wood's sympathies have always been strongly on the side of athletics as a feature of university life. He is a thorough sportsman, and has spent many summers in hunting large game in Canada, Maine, and the Rockies, and many a moose, caribou, deer, elk, antelope, and bear has fallen to his rifle.

Now that Dr. Wood has returned to his teaching after a year spent in travel and recreation, his welcome home must have been one of the most gratifying features of his life, and the students who listen to his instruction may well count themselves fortunate.

WILLIAM W. KEEN, M.D., LL.D.

AFTER a tour of the world and a respite of nearly a year and a half from the arduous life of a surgeon, Dr. Keen has returned to take up the duties of his profession. After forty years of practice, there is no evidence that he has any desire whatever to lighten the burdens which, had he remained an army surgeon, he would now have relinquished, as the regulations require.

A graduate of Brown University, he studied medicine in Jefferson Medical College, Philadelphia, graduated during the Civil War, and immediately received a commission as an Acting Assistant Surgeon. He was assigned to duty first in Washington, then to temporary field duty, after which he served in the Satterlee Hospital, Philadelphia. His best known early work, however, was done in the United States military hospital at Christian Street and later at Turner's Lane, Philadelphia, in conjunction with Drs. S. Weir Mitchell and George R. Morehouse.

It was a fortunate circumstance that associated him with men whose minds were alert to the remarkable opportunities for clinical research which the newly organized hospital for injuries and diseases of the nervous system afforded.

The wealth of material was carefully and systematically studied, and formed the basis of the publications of Mitchell, Morehouse, and Keen on "Gunshot Wounds and other Injuries of Nerves," which is the starting-point for our modern knowledge of such injuries and their treatment. Several other joint monographs also were published by these three gentlemen.

At the close of the war, after having spent two years in European medical centres, Dr. Keen established himself in Philadelphia and began at once to teach anatomy and operative surgery. For ten years he dissected and lectured in the Philadelphia School of Anatomy (founded in 1820), following D. Hayes Agnew, and continued successfully the practical courses that were so much appreciated. During this period Dr. Keen held the lectureship on pathological anatomy at the Jefferson Medical College. He became professor of the principles and practice of surgery in the Woman's Medical College in 1884, and on the death of Samuel W. Gross, in 1889, suc-

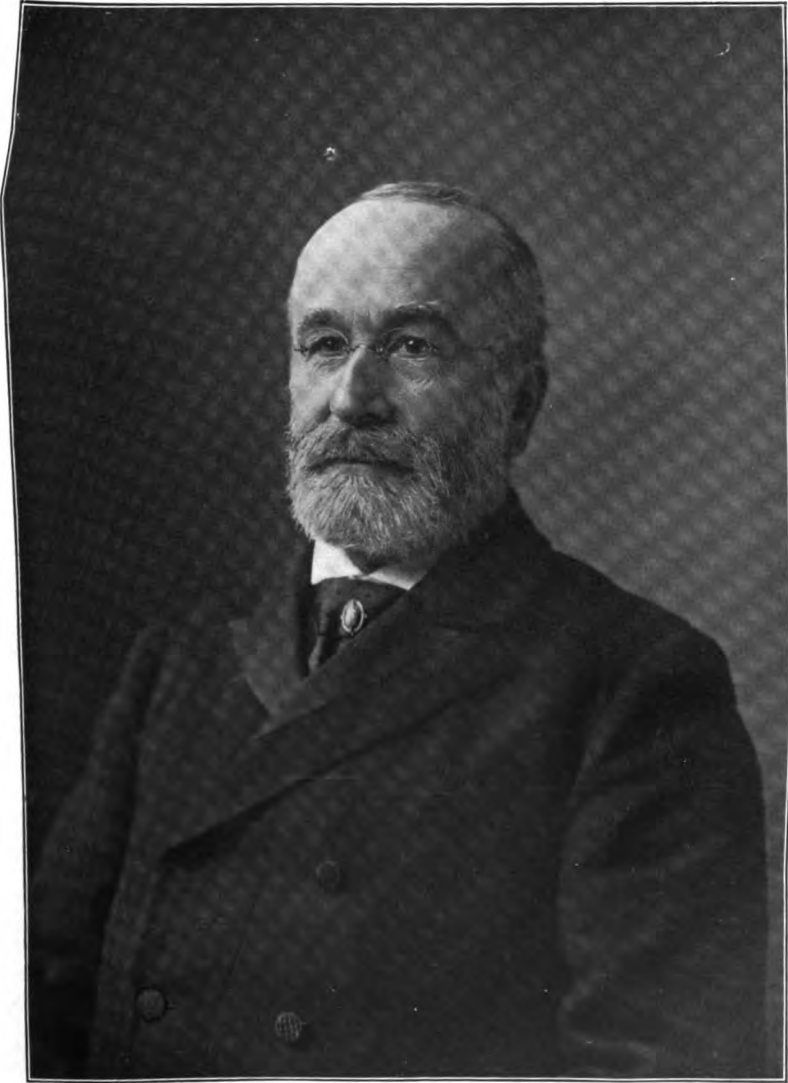


FIG. 1.—William W. Keen, M.D., LL.D.

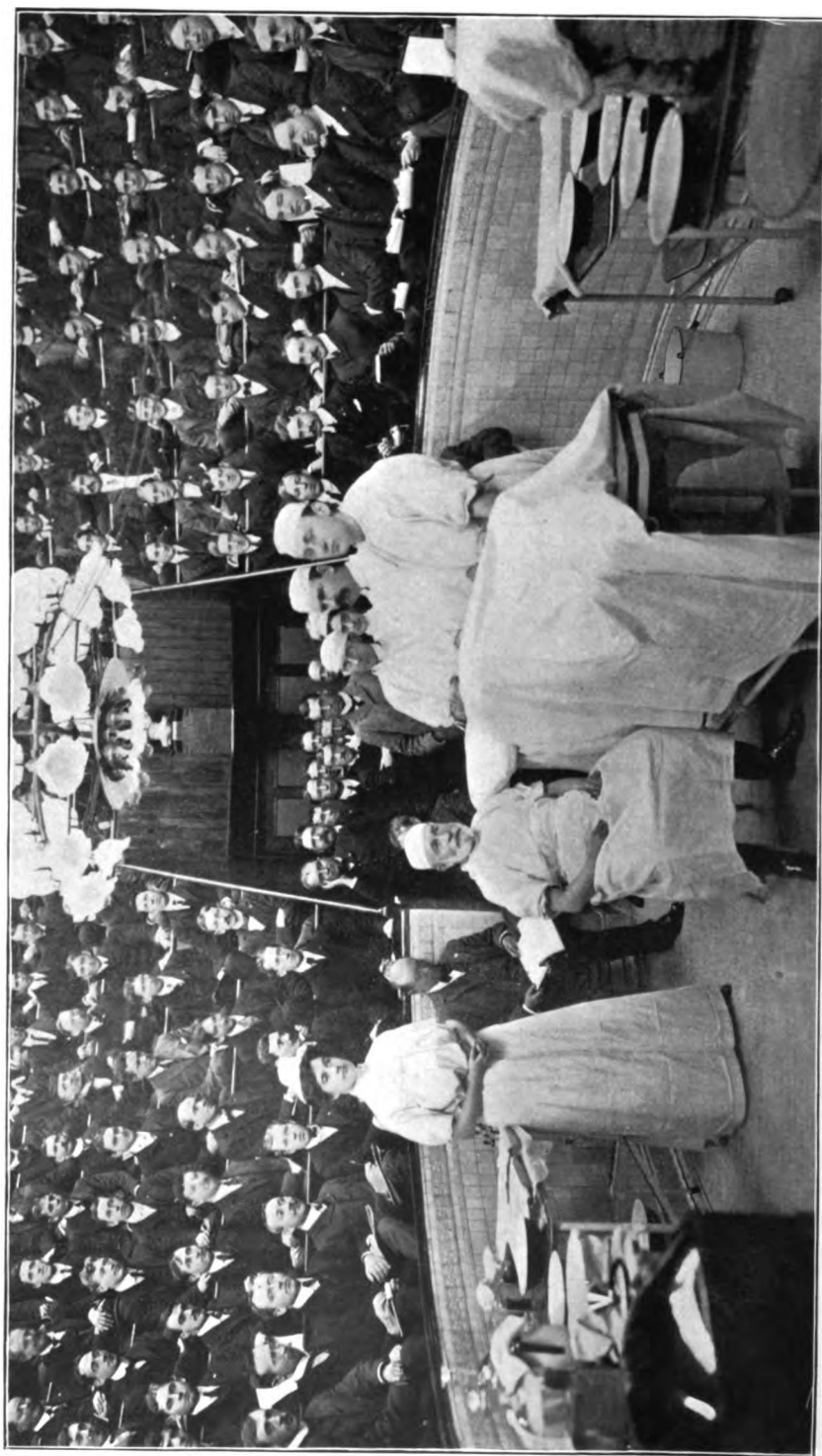


FIG. 2.—Dr. William W. Keen operating at the Jefferson Medical College.
(From a recent photograph taken especially for the CURTIS.)

ceeded him as professor of surgery in the Jefferson Medical College, a position which he has now filled for nearly fourteen years.

In two other institutions Dr. Keen has taken the greatest interest. He joined the Academy of the Natural Sciences of Philadelphia in 1867 and has maintained an active membership in that body. He also allied himself with the Pennsylvania Academy of the Fine Arts, and for thirteen years was professor of artistic anatomy. He entirely reorganized the teaching of artistic anatomy, thereby rendering an important service to a group of artists whose fundamental knowledge of anatomy insured them success and has brought, in many cases, wide distinction.

Since 1873 Dr. Keen has been, first, a Trustee, and later a Fellow of Brown University, which institution conferred upon him the degree of LL.D. in 1891.

While fulfilling the demands of an active practitioner and consultant, Dr. Keen has devoted a great deal of time to the literary side of his profession. He edited and greatly improved that noble work, Gray's "Anatomy." With Professor J. William White and others he published the "American Text-book of Surgery." Dr. Keen's contributions to surgical literature are over two hundred in number, and deal for the most part with the most difficult problems of surgical practice. His work on the "Surgical Complications and Sequels of Typhoid Fever" is one of his most important recent publications, and has been greatly appreciated by the general practitioner. In the field of brain surgery Dr. Keen stands preëminent in this country, and during the past year we have lamented the absence of his counsel and the assuring and confident manner so welcome in the midst of grave emergency.

In 1900 he was elected the president of the College of Physicians of Philadelphia, succeeding Ashhurst, Mitchell, Da Costa, Agnew, and others who for more than a century have graced the chair. Dr. Keen has been most active in his efforts to improve the efficiency of the college and add to its library. He gave five thousand dollars to the endowment of the library, and by his individual efforts succeeded in raising over fifty thousand dollars for this purpose. This money came not altogether from medical men, but very largely from public-spirited men and women whom Dr. Keen persuaded to contribute to one of the oldest and most influential medical societies in America.

In addition to corresponding membership in the Société de Chirurgie de Paris and honorary membership in the Société Belge de Chirurgie, and the Clinical Society of London, Dr. Keen has the rare distinction of being one of the eight living honorary members of the Deutsche Gesellschaft für Chirurgie.

The latest honors to be conferred on Dr. Keen are his election as President of the Sixth Triennial Congress of American Physicians and Surgeons to be held at Washington in May, 1903, and his election as Honorary President of the First Egyptian Medical Congress. There will be gathered at Washington the fourteen special societies composing a medical congress the scientific value of which is not excelled.

Dr. Keen has been honored with an election to the highest offices in the gift of the medical profession. In 1898 he was President of the American Surgical Association, and in 1899 was elected to the presidency of the American Medical Association, presiding over the largest meeting ever held by that society, the most important and comprehensive of all the medical societies in America.

At the close of the Spanish-American War President McKinley appointed Dr. Keen the medical member of the commission to investigate the conduct of the war, but owing to other pressing engagements he was obliged to decline the appointment. In 1900 he received, with three other American surgeons, the honorary fellowship of the Royal College of Surgeons of England.

Dr. Keen's honors are thus coming from the East and from the West, from the oldest nations and from the youngest, with a spontaneous expression of good-will and good-fellowship that must be highly gratifying to himself, as it surely is to all his friends.

Monograph

THE BLOOD IN HEALTH AND IN DISEASE, WITH A REVIEW OF THE RECENT IMPORTANT WORK ON THIS SUBJECT.

BY THOMAS R. BROWN, M.D.,

Instructor in Medicine, Johns Hopkins Medical School, Baltimore, Md.

(I.) INTRODUCTION.

AMONG all the advances in modern medicine, which have come from the application of the laboratory method to the diagnosis, prognosis, and treatment of disease, none has contributed more than that derived from the careful and systematic examination of the blood; in fact, so much work has been done upon this subject that it has given rise to a new subdivision in medical nomenclature, hæmatology, with a literature and terminology of its own. In discussing a subject of such magnitude, it will be obviously impossible in the limits of the present communication to consider every article which has appeared upon the blood in health or in disease; for that reason we shall take up systematically the various divisions of the subject, giving first the generally accepted views held, and follow this with a *résumé* of the articles of greatest importance and interest in that connection.

Thus we shall take up in order the consideration of (II.) constituents of the blood, taking up separately the plasma and the formed elements; (III.) methods of blood examination, including examination of fresh, dried, and stained specimens, the quantitative estimation of the various corpuscles and hæmoglobin, and the bacteriological, physical, and chemical examination of the blood, although the bacteriological study will be considered more in detail under the subject of serum diagnosis, serum therapy, and immunity; (IV.) the origin and significance of the various blood-cells and blood-

granulations, normal and pathological; (V.) the blood at different periods of life; (VI.) the influence of exposure to cold and to high altitudes upon the constitution of the blood; (VII.) the differentiation of human blood from that of animals; (VIII.) the anæmias, including primary pernicious anæmia, chlorosis, secondary anæmia, leukæmia, pseudoleukæmia, and splenic anæmia; (IX.) leucocytosis, including the physiological and the different varieties of the pathological; (X.) leucopenia; (XI.) the blood in special diseases and pathological conditions, including the blood in infectious diseases, in diseases due to animal parasites, in inflammatory processes, abscesses, appendicitis, etc., especially coming under the care of the surgeon, the blood in cardiovascular diseases, in diseases of nutrition and metabolism, in cancer, and other diseases of the gastro-intestinal tract, in mental and nervous diseases, in pregnancy, labor, and the puerperium, in diseases of the skin, in various intoxications, and in various other diseases and pathological conditions; and (XII.) serum diagnosis, serum therapy and immunity, in which division, after a review of some articles of general interest in this connection, there will be taken up in turn serum diagnosis, serum therapy, and immunity in typhoid fever, diphtheria, tuberculosis, tetanus, pneumonia, and a number of other infectious diseases, in which work along these lines has been done.

Of course, the main object in this work will not be to furnish an article which may act as a text-book, but to give a critical review of the recent work upon this subject, much of which has not as yet made its appearance in text-books. We have added the brief chapter on serum therapy, serum diagnosis, and immunity because we feel that no article on blood is complete without a consideration of these most interesting and important questions in modern medicine, although the subject is not generally considered in articles on the blood.

(II.) CONSTITUENTS OF THE BLOOD.

The blood is made up of a fluid portion or plasma and the formed elements, consisting of red blood-corpuscles or erythrocytes, white blood-corpuscles or leucocytes, blood-plaques or plates or platelets, and blood-dust.

The specific gravity of normal blood varies between 1.058 and

1.062, being higher in men, lower in women, while in the pathological conditions it may vary between 1.025 and 1.068, being decreased in nephritis, chlorosis, and cachectic conditions, as a rule, and increased in febrile diseases, affections associated with cyanosis, obstructive jaundice, and diseases associated with severe diarrhœa.

The solids in blood average in men 21.6 per cent., in women 19.8 per cent.; the reaction is alkaline, due to the presence of disodic-hydric-phosphate and sodium carbonate, the alkalinity being increased at the beginning of digestion, but decreased later, and also decreased after exercise and in the case of severe anæmias (except chlorosis), leukæmia, uræmia, hepatic diseases, diabetes, high fevers, etc.

As is well known, the functions of the blood represent the functions of its constituents, and thus it serves to carry oxygen from the lungs to the tissues, and to carry carbon dioxide and other waste products of metabolism to the lungs and the excretory organs; to carry foodstuffs to the tissues; to act as a medium for the transmission of the internal secretions of various glands; to aid in equalizing the temperature and water contents of the body; to furnish the agents designed to destroy or remove invading bacteria or other foreign substances; and to furnish the substances out of which the pigments and many of the various secretions of the body are elaborated.

(a) FLUID PORTION OR PLASMA.—The plasma or fluid portion of the blood is made up of water, various albuminous constituents, as fibrinogen, serum albumin, and serum globulin, extractives, such as urea, uric acid, creatin, glycogen, and carbamic acid, and mineral salts; although at present comparatively little work of clinical value has been done in regard to the normal and pathological changes in the plasma, nevertheless this is undoubtedly a field which is waiting for a greater refinement in the methods of physiological chemistry to furnish great aid in the diagnosis, prognosis, and therapy of disease. This subject will be discussed further under Section III., parts 6 to 9, while Section XII. is entirely given up to the relation of the blood-plasma and blood-serum to those most interesting questions in modern medicine, serum therapy, serum diagnosis, and immunity.

(b) FORMED ELEMENTS. (1) *Red blood-corpuscles*.—The red

blood-corpuscles possess, of course, the important function of carrying oxygen from the lungs to the tissues, this being brought about by the presence in the red blood-corpuscles of the substance hæmoglobin, which takes up oxygen in the lungs, forming the unstable compound oxyhæmoglobin, which in turn gives up its oxygen when brought in contact with the various organs and tissues of the body, and thus renders possible the metabolic changes necessary to the life of the cell.

From the hæmoglobin various other blood and body pigments are formed, such as hæmatin, methæmoglobin, hæmatoidin, hæmatoporphyrin, the biliary and urinary pigments, etc. The hæmatin is interesting, because by the addition of hydrochloric acid the characteristic hæmin crystals are formed, which are of value medicolegally in determining whether a suspected substance does or does not contain blood.

The red corpuscles or erythrocytes are small and, under normal conditions, biconcave disks, seven to eight micromillimetres in diameter, and number about 5,000,000 per cubic centimetre in men, and about 500,000 less in women, although these numbers vary markedly under absolutely normal conditions, 6,000,000 and over being sometimes met with in perfectly healthy individuals. The number is increased in those conditions associated with concentration of the blood, such as profuse diarrhœa, cholera, etc., while in pathological conditions associated with destruction of the red blood-corpuscles, or with the dilution of the blood by relative increase of the plasma, the number is diminished, these conditions being known respectively as polycythæmia and oligocythæmia. Under pathological conditions we may have corpuscles which show variation in size, shape, and color reaction, and also may meet with nucleated forms; thus we may have microcytes, macrocytes, poikilocytes, polychromatophilic corpuscles, and erythroblasts or nucleated red corpuscles, normoblasts, microblasts, megaloblasts, and giantoblasts.

Besides combining with oxygen the hæmoglobin can combine with carbon monoxide, nitric oxide, etc., and this is of especial interest in toxicology, as for instance in coal-gas poisoning. Hæmoglobin and all its compounds and derivatives have characteristic spectrum bands, and the spectroscopic test is frequently used in medicolegal and other work.

The study of the red blood-corpuscles is of paramount importance to the physician in the diagnosis and prognosis of the primary and secondary anæmias, of great aid to the surgeon in deciding for or against the advisability of operation, and extremely useful to the therapist in determining the value of various forms of treatment, especially as regards the administration of iron, which, as is well known, is one of the most important constituents of hæmoglobin; all these subjects will be carefully considered under the appropriate sections.

(2) *White blood-corpuscles.*—The white blood-corpuscles or leucocytes have a distinctly different function from that of the red corpuscles, for the brilliant work of Metchnikoff, Buchner, and others has shown, beyond a doubt, that one and probably the most important function of the leucocytes is phagocytosis, that is, the removal or destruction of bacterial, chemical, or mechanical irritants, or the products of bacterial activity, while recent work seems to demonstrate that the bactericidal agent in serum, lymph, and exudates is a nucleo-proteid derived from the leucocytes; the leucocytes are also undoubtedly important factors in the resorption of the products of cellular destruction and necrobiosis, in transforming these products, and in aiding in their elimination, while possibly they play an active rôle in nutrition, and may be regarded as cells filled with an alimentary reserve.

The leucocytes are nucleated, colorless corpuscles normally present from 5000 to 7000 per cubic millimetre, although in very young children they may be present in greater abundance. They are not all of one kind, but may be divided into: the small and large mononuclear forms, the former of which are frequently known also as lymphocytes, these cells possessing a round or oval nucleus and a protoplasm, which is not granular; polymorphonuclear granular leucocytes, of which we have the finely granular or neutrophilic and coarsely granular or eosinophilic, while basophilic leucocytes are found in very small proportion in normal blood, although increased in certain pathological conditions, especially myelogenous leukæmia; transitional leucocytes, in which the nucleus is slightly indented, and the protoplasm somewhat granular, while Cabot believes that the myelocytes or mononuclear neutrophils are found in small quantities in normal blood. The percentage of the various forms of leucocytes is given about as follows:

small mononuclears fifteen to thirty per cent.; large mononuclear and transitional forms four to eight per cent.; polymorphonuclear neutrophiles sixty to eighty per cent., and eosinophiles one to five per cent. From a series of observations made by us upon fifty normal adults we found that the average number of leucocytes per cubic millimetre was 6500, of which seventy to seventy-four per cent. were polymorphonuclear neutrophiles, eighteen to twenty-two per cent. lymphocytes, six to seven per cent. large mononuclear and transitional cells, and one to two per cent. eosinophiles.

The changes in the total number of white blood-cells per cubic millimetre and the relative changes in percentage of the various forms will be carefully considered under the subject of leucocytosis, leucopenia, and the changes of the blood in special diseases and pathological conditions.

(3) *Blood-plates*.—These small, round, colorless bodies one and one-half to three and one-half micromillimetres in diameter are found in normal blood regularly, the number present per cubic millimetre being given from 180,000 upward to 300,000 by different observers. Their pathology is but little known beyond the probability that they play an important rôle in blood coagulation and in thrombus formation.

(4) *Blood-dust*.—Müller described in 1896 small, round, colorless granules from one-fourth to one micromillimetre in diameter, which he called hæmoconien, and which Stokes believes to be the extruded granules of neutrophilic and eosinophilic leucocytes. Up to the present, no diagnostic or prognostic value has been ascribed to these bodies, although Stokes thinks it possible that they may bear some relation to the immunizing properties of the serum.

(III.) METHODS OF BLOOD EXAMINATION.

(a) EXAMINATION OF FRESH BLOOD SPECIMENS.

The examination of a fresh specimen of blood should be made in every doubtful case which comes either to the physician or to the surgeon; in fact, it has seemed to us that this should be one of the routine procedures in every case, for it takes but a slide and cover-slip, a moment's time and ability to use a microscope, and is fraught with great advantage in diagnosis both positive and negative. It tells us in a broad way whether the red blood-corpuscles are normal in size and shape, or whether they show varia-

tions in size, shape, or color contents, that is, evidences of anæmia; whether the leucocytes are present in approximately normal numbers, whether there is a distinct increase or decrease, or whether the relative number of any one form is especially increased or decreased; whether leucocytes are present which are not met with normally; whether the blood contains parasites, and the variety of parasites present, and whether the deposition of fibrin and the tendency towards rouleaux formation of the red blood-corpuscles is normal or abnormal. The method of fresh blood examination is especially useful in the diagnosis of malaria, for by no other means can the variety, size, age, and general characteristics of the various forms of malarial parasites be so satisfactorily demonstrated.

(b) EXAMINATION OF DRIED AND STAINED SPECIMENS.

The study of the blood has been completely revolutionized by the introduction of the differential methods of staining, as suggested by Ehrlich in 1878. His method, based upon the fact that certain granulations possess affinity for certain stains, has been followed by practically all other students along this line. After making the blood smears on the cover-slips, it is necessary to fix them before adding the stain. The fixing agents which have proven most satisfactory are: heat, either passing the cover-slips several times through the flame or heating at 110° C. for one hour, which is especially applicable to the study of the leucocytes which are to be stained by the Ehrlich stain; absolute alcohol and ether, which are of especial value in the study of malarial parasites; formalin and chromic acid. For ordinary purposes, passing the cover-slips several times through the flame, as recommended by Kanthack, is probably the most satisfactory, as it requires but little time, and renders the leucocytes especially easy of study. As to the stain to be used, this, of course, depends somewhat upon the especial constituents of the blood which we are studying in the special cases. Thus, if the leucocytes are to be especially studied and differential counts made of the various forms, no stain is comparable to the Ehrlich triple stain or its modifications. The Jenner stain is also useful, especially as it fixes and stains at the same time. As a stain for the malarial parasites, eosin and methylene blue, or the Chenzinsky-Plehn solution of methylene blue, or the Fletcher

and Lazear stain of thionin, or the Romanowsky stain, is to be advised. For basophilic granules, the most satisfactory is Dahlia, while for glycogen granules a solution of iodine in potassium iodide and water is to be recommended.

Besides these more or less stock methods of staining, a number of interesting new stains have been suggested recently. Knijaskow¹ recommends as a fixing agent a mixture of equal parts of one per cent. aqueous solution of osmic acid and two per cent. alcoholic solution of bichloride of mercury, leaving the blood-films in the mixture three minutes, then dropping them in distilled water containing a few drops of acetic acid, and then washing in water. He recommends as stain for the blood-plates methylene blue and five per cent. carbol fuchsin.

Michaelis² describes a universal coloring method for blood preparations, this preparation staining at the same time neutrophiles, eosinophiles, basophiles, and blood-platelets. The blood is fixed by absolute alcohol or by heat. Two preparations are used: (1) one per cent. aqueous solution of methylene blue (absolutely pure), and (2) one per cent. aqueous solution of eosin, great care being taken that distilled water is used. From these are prepared two other solutions: (*a*) twenty cubic centimetres of stock solution No. 1 and twenty cubic centimetres of absolute alcohol, and (*b*) twelve cubic centimetres of stock solution No. 2 and twenty-eight cubic centimetres of acetone. To stain, one mixes one cubic centimetre each of solutions (*a*) and (*b*), pours the mixture into a glass dish, in which the blood preparation is placed, smear side down, and the dish is kept carefully covered. The time required for staining varies between one-half minute and ten minutes. Hewes³ recommends that after the specimen has been stained by Ehrlich tri-acid stain, it should be washed in water and then stained from one-half second to ten seconds in Löffler's alkaline methylene-blue solution.

May and Grünwald⁴ recommend the use of the following stains:

¹ Centralbl. f. allg. Path. u. path. Anat., 1899, p. 398.

² Deutsche med. Wehnschr., 1899, No. 30.

³ Boston M. and S. J., July 13, 1899.

⁴ Centralbl. f. innere Med., Mar. 15, 1902.

One litre of one per cent. eosin is mixed with one litre of one per cent. methylene blue, and after a few days filtered by means of suction, and the precipitate washed with cold distilled water until the filtrate is almost colorless. A saturated solution of this precipitate in methyl alcohol is the coloring agent they recommend, the blood-films not being fixed before being put into this solution. The blood-films are left in this solution for about two minutes, as a rule, after which they are washed in distilled water, to which a few drops of this solution have been added. The various cells are stained as follows:

The erythrocytes bright red, the nuclei of the leucocytes deep blue, the alpha-granules deep red, the epsilon-granules deep blue, the gamma-granules bright red, while pathological granules, malarial plasmodia, and bacteria are well stained by this means.

A stain which seems to promise much is that described by Wright.¹ Fixing and staining are done by a specially prepared combination of eosin and methylene blue; it is especially satisfactory for the study of malarial parasites and blood-platelets.

Ito² recommends the following method for the staining of the blood, which he has used in Senator's clinic. The blood is spread in a thin layer upon a cover-slip, upon which previously an alcoholic staining solution has been applied; this is then placed upon a slide with a depression in its centre (such as is used for studying the hanging drop), and in this depression a drop of water is placed. By this means he has studied the blood under various conditions and with various stains.

Rosin and Bibergeil³ have performed some most interesting experiments upon vital blood staining, in which methylene blue, neutral red, eosin, and a few combinations of these were used. They found Ehrlich's basophilic granules to be normal constituents of the erythrocytes, although pathologically they were increased, while with the use of acid coloring agents, the phenomenon of the ante-mortem movements of the granules of the amœboid elements was met with. The article is of especial interest in connection with the possibilities of its further application in the study of other tissues and organs.

¹ J. of Exper. Research, Jan., 1902.

² Allg. med. Centr. Ztg., 1901, No. 101.

³ Deutsche med. Wchnschr., 1902, Nos. 3 and 4.

(c) ESTIMATION OF THE RED CORPUSCLES.

The most satisfactory method of counting the red blood-corpuscles is that devised by Thoma, in which the blood is diluted either with Toisson's solution, Hayem's fluid, or Pacini's fluid, and then counted by means of the counting chamber prepared by Zeiss, by Leitz, and by other manufacturers. The tintometer of Oliver is of value in counting the red blood-corpuscles when the number is not much below normal, but it has not proven quite so exact in severe cases of anæmia, especially pernicious anæmia, while Daland's hæmatocrit furnishes a rapid method for estimating approximately the number of red blood-corpuscles.

(d) ESTIMATION OF THE HÆMOGLOBIN.

The estimation of the hæmoglobin is equally as important as the determining of the red corpuscles, because in the majority of anæmic conditions the relative diminution in hæmoglobin and in red corpuscles distinctly differs; thus, in the majority of secondary anæmias and in chlorosis, the red corpuscles are relatively less reduced than the hæmoglobin; in other words, the color-index is low, and fresh, and stained specimens of red corpuscles show distinct pallor, while in many cases there is a tendency towards the presence of many microcytes. In pernicious anæmia, on the other hand, the relative diminution of red corpuscles is greater than that of the hæmoglobin; in other words, the color-index is high, and in the fresh and stained specimens, the corpuscles, as a rule, show no pallor, in fact, a deeper color than normal, while very many megalocytes are present. The instruments used for determining the hæmoglobin are many, but those most satisfactory are Von Fleischl's instrument, although it is not so exact as the Miescher modification, the Oliver hæmoglobinometer, the Tallqvist hæmoglobin scale, which is the cheapest and simplest of all instruments and gives approximately correct results, and the Dare hæmoglobinometer,¹ which differs from the others in the fact that the blood is not diluted. The Gower instrument is not so exact, and has been given up by many. In our hands the most satisfactory results have been obtained with the Dare and with the Von Fleischl

¹ Phila. M. J., Sept. 22, 1900.

instruments, although the Tallqvist scale has proved very satisfactory in cases in which only the approximate amount of hæmoglobin is desired.

(e) ESTIMATION OF THE WHITE CORPUSCLES.

The estimation of the white corpuscles is made practically in the same way as that of the red, that is, the blood is diluted and then counted in the Zeiss or Leitz instrument. The best diluting fluid is a solution of acetic acid, between one-third and one per cent., while if both red and whites are to be counted at the same time, Toisson's solution is used, as the acetic acid dissolves the red corpuscles. Although an especial pipette for the counting of the leucocytes is furnished in the blood-counting cases, in which the blood is diluted one to ten, nevertheless most clinicians prefer to count both red and white corpuscles with the red pipette.

(f) ESTIMATION OF THE BLOOD-PLATES.

Determann¹ gives the following method for counting and staining the blood-plates.

The diluting fluid used is a nine per cent. solution of sodium chloride, to which a little methylviolet has been added, or a mixture of one per cent. solution of sodium chloride and five per cent. solution of potassium bichromate; a drop of either of these is placed on the finger and through this the finger is pricked. The blood and fluid are mixed with a cover-glass and then transferred to a Thoma-Zeiss counter. The relation of the blood-plates to the red blood-cells was found by this method to be: average of twenty-five cases, one to twenty-two; maximum, one to eighteen; minimum, one to thirty. An increase of the blood-plates was noted in many diseases associated with anæmia. For obtaining stained specimens the blood is mixed with either of the above diluting fluids, and the films made from this stained with methylviolet.

(g) ESTIMATION OF THE COAGULATION-TIME.

The determining of the coagulation-time is of practical value, especially to the surgeon in determining whether or not the subject brought to him for operation is in a condition to be treated.

¹ Deutsche Arch. f. klin. Med., vol. lxi., Nos. 3 and 4.

In a certain number of diseases, the coagulation-time is markedly increased, the blood taking from six to twenty minutes to coagulate, instead of the normal from two to four minutes; obviously, in such cases, death from capillary hemorrhage might be likely to follow a surgical operation. The most important of such conditions are diseases associated with jaundice, hæmophilia, purpura, and allied conditions. The method of determining the coagulation-time is by means of the tubes devised by Wright, in which the blood is sucked up and its fluidity tested from time to time, generally at intervals of one-half minute.

(h) BACTERIOLOGICAL STUDY OF THE BLOOD.

Within recent years, many attempts have been made to study the blood bacteriologically in a number of diseases, notably, typhoid fever, pneumonia, and septicæmia. The method applied by the more recent investigators, as Prochaska, Cole, and Busquet, has been the removal of a considerable quantity of blood from the veins in the anterior aspect of the elbow, under the strictest aseptic precautions, and making cultures of the blood so obtained upon the various media. This has led to the diagnosis being made in cases of typhoid fever before the appearance of the Widal reaction, and in various cases of pneumococcus infection. The subject will be considered at length under Section XII.

(i) SERUM REACTION.

The amount of work done within the past few years upon the subject of serum diagnosis has been so enormous that we shall not attempt to discuss the subject here, but have devoted the whole of Section XII. to a discussion of serum diagnosis, serum therapy, and immunity.

(j) PHYSICAL AND CHEMICAL EXAMINATION OF THE BLOOD.

The physical and chemical examination of the blood up to the present time has been made largely by physiologists, and has furnished comparatively little of value to the clinician; nevertheless, all students in this field feel sure that, in a comparatively short time, this will be one of the most important sub-divisions of blood examination, when greater refinement in the bio-chemical methods is established. The subject of the alkalinity of the blood has been

studied with great care of late. Hladik¹ recommends Behrend's method: one cubic centimetre of blood is centrifugalized with five cubic centimetres of one per cent. sodium-chloride solution, and first the blood-serum, later the red blood-corpuscles, dissolved in water and titrated with a one to fifty normal acid solution, litmus being used as an indicator. He thinks this gives a truer reading than the determination of the alkalinity by examination of the ash, as the two non-saturated sodium phosphates, by heating in the presence of sodium chloride, increase in alkalinity.

Burmin,² using a slight modification of Landois's method, determined the alkalinity in a number of pathological conditions. He found a diminished alkalinity in cirrhosis of the liver, jaundice, phthisis, nephritis, malaria, leukæmia, anæmia, diabetes, gout, and obesity. He also found a decrease in alkalinity in nine cases of simple chlorosis, although other observers have described an increased alkalinity in this condition. Burmin found that in these nine cases, synchronously with the administration of iron, there arose a gradual increase in alkalinity. A transitory increase in alkalinity could be brought about by drinking alkaline waters, as Vichy.

Fodera and Ragona,³ from experiments upon rabbits and dogs, conclude that the alkalinity of the blood varies with the food; that it is diminished after fasting, after the ingestion of hydrochloric acid, and after slowly produced asphyxia, while it is increased after the administration of alkalies by the mouth. They were unable to show any relationship between destruction of red blood-corpuscles and alkalinity of the blood. The indirect method of determining the percentage of hæmoglobin in the blood by the determination of the specific gravity of the blood is coming into greater and greater favor, the Hammerschlag method being usually employed.

Van Spanje⁴ recommends a slight modification of the usual method for determining the specific gravity of the blood. He uses a mixture of chloroform and olive oil in the proportion of three

¹ *Ztschr. f. klin. Med.*, vol. xxxix. p. 194.

² *Ibid.*, vol. xxxix. p. 365.

³ *Arch. ital. de biol.*, vol. xxix. 1. p. 34.

⁴ *Nederl. Tijdschr. v. Genees.*, 1900, vol. i. p. 1107.

parts of chloroform, and one part of oil; this gives a mixture of the specific gravity 1.056. He recommends this because it is extremely easy to prepare and is less affected by atmospheric conditions than the usual chloroform-benzol mixture.

The determination of the sugar in the blood, normally present in from one to one and one-half parts per thousand, is of importance, in the first place, in diabetes where it frequently reaches nine parts per thousand, and, in the second place, in differentiating carcinoma from sarcoma; in the former condition, an increase of sugar is almost constantly met with, while it is not in the latter. The diabetic blood-test of Williamson is based upon this increase of sugar in the blood in diabetes. The method of making the test is as follows:

Twenty cubic millimetres of blood are added to forty cubic millimetres of distilled water, and to this one cubic centimetre of one-six-hundredth aqueous solution of methylene blue and forty cubic millimetres of six per cent. aqueous solution of potassium hydroxide; in the case of normal blood, after being kept in the thermostat for an hour, or in boiling water for from three to four minutes, there is no change in color, while in the case of diabetic blood the blue changes to a dirty green.

Among other interesting chemical tests for the blood may be mentioned that for glycogen, which, as a rule, is increased in pneumonia, in typhoid fever, in phthisis, in diabetes, and in leukæmia, decreased in hepatic and cardiac diseases associated with fever, and absent in acute rheumatism; in fat, and the fatty acids which are increased in obesity, after eating fat, in chronic alcoholism, in diseases affecting the long bones, in severe cases of diabetes, in chronic nephritis and tuberculosis; in the various biliary pigments which are found in the blood in obstructive jaundice, and in diseases associated with marked destruction of blood-corpuscles.

Engelhardt¹ has determined in a most careful way the quantity of fat present in the blood of normal individuals and of those suffering with diseases associated with severe cachexia. The average in eight healthy individuals was 0.194 per cent., while in five very cachectic individuals it was 0.174 per cent. His results,

¹ Deutsches Arch. f. klin. Med., vol. lxx., Nos. 1 and 2.

therefore, are opposed to those of Rosenfeld, who thought that there was regularly an increase of fat in cachectic individuals.

Viola¹ has tested the electrical conductivity and the changes in the freezing point of the blood-serum under various conditions. In chronic serous pleurisy the conductivity was normal or subnormal in all cases except one; the cases of chronic nephritis, however, with or without uræmic manifestations, almost always show distinct increase.

The freezing point of the normal blood-serum varies, according to Viola, between 0.54° C. and 0.59° C. Between electrical conductivity on the one hand and the freezing-point and sodium-chloride contents of the blood-serum on the other hand, no constant relationship can be shown.

Haldane and Smith,² from a carefully performed series of experiments, conclude that the generally accepted view that the blood constitutes from one-twelfth to one-thirteenth of the body weight is incorrect; according to them the average mass of blood is 4.9 per cent. of the body weight. The oxygen capacity of the blood in litres averages 0.85 per cent. of the body weight in kilogrammes. The percentage of oxygen in the blood averages 18.5 per cent., and the oxygen capacity of the blood of men and various animals is directly proportional to the color contents of the blood.

(IV.) THE ORIGIN AND SIGNIFICANCE OF THE VARIOUS BLOOD-CELLS AND BLOOD-GRANULATIONS, NORMAL AND PATHOLOGICAL.

(a) RED BLOOD-CELLS AND GRANULATIONS IN THE RED BLOOD-CELL.

The view which is generally held regarding the origin of the blood-cells in postembryonic life is that they are derived from nucleated blood-corpuscles, mainly, if not exclusively, in the bone-marrow, either by a disappearance or extrusion of the nucleus.

During the past few years several articles of value have appeared concerning this question. Engel³ has studied the embryonic blood, in the various stages of the development of the reds, in human beings and dogs, the preparations being fixed by heat and

¹ Riv. veneta di sc. med., 1901, No. 8.

² J. Physiol., 1900, vol. xxv. p. 331.

³ Deutsche med. Wehnschr., 1899, No. 13.

stained with Ehrlich's triacid stain. From these studies he concludes that the bone-marrow is the seat of origin of the blood-cells, and that the non-nucleated cells are derived from the nucleated red blood-cells, mostly by disappearance and partly by extrusion of the nucleus. He agrees with Ehrlich that the presence of megaloblasts in pernicious anæmia is an extremely unfavorable sign, and also that from the study of the blood alone pernicious anæmia can be diagnosed. According to Junger¹ the fate of the nucleus of the nucleated red corpuscles in the bone-marrow appears to be a shrinking and diminished ability to take the stain; the nucleus then assumes a peripheral position and may break up into small parts which may be separated or united. There is then a moderate karyolysis, and finally the nucleus disappears entirely.

The blood-forming properties of bone-marrow have been shown by many observers, as Cohnheim, Bizzozero, Golgi, Neumann, Salvioli, Litten, and Domenici. Recently Zen² has devoted considerable time and attention to this subject. His work comprised sixty-four cases in all, the ages varying from eight to eighty-eight years. To determine whether the bone-marrow was in active function, he used as a criterion the presence of nucleated red blood-cells in the circulating blood. He concludes that in health the blood-forming property of the marrow of the long bones is resting, as also in the cases of infectious diseases of short duration (as pneumonia). On the other hand, in infections of longer duration, as typhoid, the blood-forming function is present, but does not begin before the end of the second week. The age of the patient seems to be without influence upon the blood-forming properties of the bone-marrow. Kolliker and Ecker were the first to call attention to the spleen as a blood-destroying organ. Reich³ has carried on an investigation in regard to this point upon the frog's spleen, and comes to the same conclusion, showing all forms of degeneration of the red blood-cells. First, defects and vacuoles containing hæmosiderin-granules made their appearance in the cell's periphery; the process gradually became more and more marked, until finally the whole cell was affected. The nucleus also undergoes changes; it

¹ *Deutsches Arch. f. klin. Med.*, 1900, vol. lxxvii., Nos. 1 and 2.

² *Gazz. d. osp.*, 1900, No. 63.

³ *Fortschr. d. Med.*, 1899, vol. xvii. p. 361.

shrinks, loses its structure, and karyolytic and karyorhexic processes set in.

As to the pathological granulations in red blood-corpuscles, the most work has been done in connection with the appearance of basophilic granulations in the red corpuscles in various diseases. Thus Hamel,¹ staining the blood with methylene blue for a considerable period of time, concludes that these basophilic granulations seem to occur most frequently in lead poisoning, in fact, are among the early signs of this disease. Among other pathological conditions, he found them in one case of pernicious anæmia, in one out of twelve cases of chlorosis, and in only the profoundly cachectic conditions in carcinoma, while they were absent in nine cases of tuberculosis, twenty-seven cases of acute febrile disease, twenty-four cases of syphilis, and twenty-six patients suffering from various other diseases. Kohn² believes these basophilic granulations to be the evidence of the partial degeneration of the protoplasm of the cell depending upon the hydræmia following the loss of blood, as he could cause them to appear after the abstraction of blood, while Litten³ found them in all cases of primary and secondary anæmia, the granulations occurring in greater number as the anæmia became worse. These granulations would, therefore, seem to be due to the anæmic degeneration of the protoplasm of the red cells or to nuclear degeneration.

Grawitz⁴ believes that these basophilic granules are simply indications of a degenerative change due to various causes, and are in no wise specific of any one affection. They were found by him to be especially numerous in pernicious anæmia, in cases with much toxic absorption, in leukæmia, and in lead poisoning. They were entirely absent in chlorosis (twelve cases), in syphilis (twenty-one cases), in advanced tuberculosis, as well as in afebrile cases, and those without cavity formation (thirteen cases), and were never found in parenchymatous nephritis, contracted kidney, or hepatic cirrhosis. The increase or decrease of these granules thus furnishes a good indication of the changes going on in certain diseases as to whether they are progressing favorably or unfavorably.

¹ Deutsches Arch. f. klin. Med., vol. lxxvii., Nos. 3 and 4.

² München. med. Wehnschr., Feb. 6, 1900.

³ Deutsche med. Wehnschr., Nov. 2, 1899.

⁴ Berl. klin. Wehnschr., Feb. 19, 1900.

Stengel, White, and Pepper¹ contribute an interesting article on the granular degeneration of the erythrocyte, in which they conclude that the granulated red blood-cells never show remains or suggestions of a former nucleus, and believe that they are due to destructive changes in the erythrocyte of the peripheral circulation rather than in the nucleated red blood-corpuscles in the blood-forming organ. One of the great arguments in favor of this view is the early appearance of these granules in cases of lead poisoning.

(b) WHITE BLOOD-CELLS AND GRANULATIONS IN THE WHITE BLOOD-CELLS.

A great deal of interest in the study of the blood in disease has centred about the question of the origin and significance of the white cells and their granulations, in the hope that these investigations might throw light upon the changes of these cells in various diseases. That the leucocyte is an important cell in the animal economy seems probable from the fact that it is met with even in the very low forms of animal life, where its functions and characteristics seem to be almost identical with those in the higher members of the animal kingdom. As to the mode of origin of these cells, there is a great difference of opinion. Ehrlich believes that the lymphocytes are formed in the lymph-glands, the neutrophiles and eosinophiles in the bone-marrow from pre-existing neutrophilic and eosinophilic cells, while the spleen is normally an organ of hæmatolysis and not hæmatogenesis. Uskoff, on the other hand, believes that all white blood-cells are derived from one form, the lymphocyte, and Gulland, who has the same belief, adds that the especial kind found in each locality depends upon the surroundings and conditions in which the cells live, a view to which Pfeiffer subscribes, basing his conclusions that the granulations are not specific, upon his finding that the same granule may be present in different types of cells, and that different granules may be present in the same cell. Koroboff,² on the other hand, has studied the part played by the spleen in blood formation, by the extirpation of this organ in dogs and by noting its effects upon the constitution of the

¹ Am. J. M. Sc., May, 1902.

² Arch. de. sc. biol., St.-Petersb., 1899, vol. vii. p. 387.

blood. According to him, the spleen must be considered as the chief organ of formation of the young elements of the blood (lymphocytes), while the other lymphatic organs play only a secondary rôle. The recent work of Pappenheim has led him to believe that the lymphocytes are derived from pre-existing cells of similar character in the bone-marrow, and he has recently¹ introduced a new nomenclature for the lymphocytes. According to the staining reaction of the protoplasm, two great groups are to be distinguished: granule-free lymphocytes and granular granulocytes, the latter being subdivided into eosinophiles, neutrophiles, etc. According to the staining reaction of the nucleus we have two groups, the amblychromatic and trachychromatic, which in the group of granulocytes are known as myelocytes and leucocytes respectively, and in the group of lymphocytes as macro-lymphocytes and micro-lymphocytes respectively.

Muir² attempts to show that the ordinary form of leucocytosis is caused by a proliferation of the bone-marrow cells, the experiments being made as follows: An experimental leucocytosis was produced in animals and, during its height, sections and smear preparations made from the marrow. According to Muir, during a marked leucocytosis, the number of neutrophilic cells in the marrow is markedly diminished, while the myelocytes themselves show marked mitotic changes. According to Muir, leucopenia is due to the heaping up of the white corpuscles in the blood-vessels of the intestines and peritoneum.

Kanthack and Hardy believe that the lymphocytes are formed in the lymphoid tissues, the neutrophiles in the intestinal tract, and the eosinophiles in the serous membranes, while Michaelis and Wolff³ distinguished two kinds of lymphoid cells, those capable of further differentiation, as the lymphoid cells of the bone-marrow, from which it is probable myelocytes may be formed, and those not capable of further differentiation, Ehrlich's lymphocytes.

Naegeli⁴ also believes that the non-granular mononuclear cells of the marrow are entirely different from lymphocytes, calling them myeloblasts, as he thinks the myelocytes are derived from them.

¹ Virchow's Arch., vol. clix., No. 1.

² J. Path. and Bacteriol., Feb., 1901.

³ Deutsche med. Wehnschr., 1901, No. 38.

⁴ Ibid., May 3, 1900.

and he believes that these myeloblasts are found in pernicious anæmia.

Lengemann¹ furnishes an interesting research upon the origin of leucocytosis. After intraperitoneal injections of liver or kidney broth, infections with staphylococci or colon bacilli, or intoxication with sodium cantharidinate, the bone-marrow becomes almost fluid and very dark in color. The leucocytes are found to be more numerous here than elsewhere in the blood. Some are seen projecting slightly into the blood-vessels, others only slightly attached to the parenchyma, while others are seen free in the current. As the marrow becomes dark red and soft, these spaces where the leucocytes are become much larger, and the leucocytes are swept away by the increased volume of blood, and appear as a pronounced leucocytosis in the blood as a whole. The hyperæmia gradually subsides, and by the eighth day the marrow contains less blood than in normal conditions, the blood being crowded out of the spaces mentioned above because of the greatly increased mitosis of the cells of the parenchyma, leucocytes, etc. This research seems to show that the marrow is involved in more affections than is usually supposed, and that the processes accompanied by leucocytosis induce marked changes in the marrow.

Schuhmacher,² by counting the leucocytes in the small veins and small arteries of the lymph-glands, concludes that there is normally a production of leucocytes taking place here.

As to the origin of the eosinophiles, various views are held, the most important of which are Ehrlich's theory that they are derived from pre-existing eosinophiles in the bone-marrow, Müller and Rieder's theory that they are derived from the eosinophiles in the circulating blood, and various theories of a local origin of these cells, from fixed connective tissue-cells in the intestines and serous cavities, from plasma-cells, from the eosinophiles normally in the tissue, and from polymorphonuclear neutrophiles in various tissues of the body, as in the bronchial mucosa in bronchial asthma, and the muscle in trichinosis.

As to the granules in the leucocytes, the work of Ehrlich upon this subject has, of course, been the most important, although, as

¹ Deutsche med. Wehnschr., Dec. 28, 1899.

² Arch. f. mikr. Anat., 1899, vol. liv. p. 331.

we have said before, his views regarding the specificity of cell-granules have been opposed by many investigators of late. Various observers have recently described pathological cell granulations; thus Hofbauer¹ found that the white blood-cells contain certain iodophilic granules almost constantly in pernicious anæmia and leukæmia, while they are absent in pseudoleukæmia and chlorosis, and scarcely present in moderate grades of secondary anæmia. The stain he used consisted of one part of iodine, three parts of potassium iodide, one hundred parts of water. According to Biffi,² the intra- and extra-cellular iodophilic leucocytic granules correspond absolutely to the eosinophile and pseudoeosinophile granules and, according to Biffi, are not glycogen but close derivatives of hæmoglobin, and are to be regarded as manifestations of phagocytosis.

According to Galli,³ the iodophile reaction possesses no clinical value, as it is met with in both normal and pathological conditions, and bears no relation to the intensity or severity of the disease.

Grünwald⁴ describes certain granules which he calls hypeosinophilic, and which he found in the mono- and polymorphonuclear cells of the blood as well as in the cells of sputum and pus. They are very fine, are stained with eosin, but decolorized by acids and alkalies, while in Ehrlich's triacid stain they appear a fuchsin red. As to the Neusser perinuclear basophilic granules, regarded by that investigator as the sign of the uric-acid diathesis, Fletcher, Simon,⁵ and several others were unable to confirm this relationship, and they also show beyond a doubt that these granules are present in a great variety of other conditions.

(V.) THE BLOOD AT DIFFERENT PERIODS OF LIFE.

As we have said before, the number of leucocytes, the number of red blood-corpuscles, and amount of hæmoglobin, are generally greater in the new-born than shortly afterwards. Schiff⁶ examined the blood twice daily from the first to the tenth day of life in fifty-eight children, determining the specific gravity by the pyknometric

¹ Centralbl. f. innere Med., Feb. 10, 1900.

² Policlin., 1901, No. 44.

³ Ibid.

⁴ Centralbl. f. innere Med., 1899, No. 30, and 1900, No. 14.

⁵ Am. J. M. Sc., Feb., 1900.

⁶ Jahrb. f. Kinderh., vol. liv. p. 1.

method. From the first to the sixth day the specific gravity varied from 1.080 to 1.070, from the sixth to the tenth day from 1.070 to 1.060; that is, the specific gravity gradually decreased from the first to the tenth day. It was higher in strong than in weak children and in those in whom the umbilical cord was tied late; it was lower in children with jaundice.

Schwinge¹ used the Thoma-Zeiss and Von Fleischl instruments in comparing the blood at different periods of life, and came to the following conclusions: The red blood-cells, hæmoglobin, and white blood-cells differ in different parts of one's life, the red blood-cells and hæmoglobin being greatest immediately after birth, shortly afterwards sinking to a minimum, and then increasing with age, with periodic fluctuations at the time of puberty, again to decrease towards the end of life, while the white blood-corpuses, on the other hand, steadily decrease in number from the time of birth to puberty, later increasing again. As to sex, the number during puberty is less in women than in men.

Carstenjen's² results regarding the relative number of white blood-cells in human beings at various ages under normal conditions are as follows: The polymorphonuclear leucocytes immediately after birth and during the first twenty-four hours show a high percentage (73.45 per cent.); they then diminish rapidly to 36.12 per cent. at the ninth day, and remain about the same during the first half year of life; they then gradually increase again to the fifth year, and between that age and the end of life vary between 51.86 per cent. and 69.22 per cent.

The lymphocytes are fewer the first day and increase to the twelfth day; from the end of the first half-year of life their percentage gradually decreases, and after the fifth year keeps fairly constant within certain limits.

The relative number of the transitional forms increases rapidly during the first week, decreases from then to the end of the first half-year, after which they remain between 6.75 per cent. and 9.47 per cent.

The number of the large mononuclear leucocytes and the eosinophiles is independent of the age of the individual.

¹ Arch. f. Physiol., vol. lxxiii., Nos. 7 and 8, p. 290.

² Jahrb. f. Kinderh., vol. lii., Nos. 2 to 4.

(VI.) THE INFLUENCE OF (a) EXPOSURE TO COLD AND HEAT AND OF (b) HIGH ALTITUDES UPON THE CONSTITUTION OF THE BLOOD.

(a) EXPOSURE TO COLD AND HEAT.

Reineboth¹ and Reineboth and Kohlhardt,² from experiments upon rabbits, exposing them in cold (2° C.) water for five minutes and estimating the hæmoglobin and blood-cells at frequent intervals, found that in all cases there was a diminution in hæmoglobin, red blood-cells and white blood-cells, lasting several days. Successive immersions always again produced a considerable decrease. Spectroscopically it was shown that there was a tendency to hæmoglobinæmia.

Grawitz³ combats these views, saying that Reineboth's and Kohlhardt's mistake was in taking the blood from veins in which the circulation was slowed by constriction. Grawitz repeated his earlier experiments, dropping rabbits into ice-water, and then taking the blood from the unconstricted veins, and was unable to detect any evidence of hæmoglobinæmia, although there was an increase in the specific gravity of the blood, which he regarded as being due to a transudation of fluid from the capillaries. Much of the effect of the cold bath followed by the warm douche Grawitz believes to be due to this exudation of serum into the tissues and subsequent return into the vessels by the application of warmth.

Hannes⁴ examined the blood of twenty-nine children in whom a marked perspiration had been produced by natural means. In seventy-seven per cent. of these a distinct leucocytosis followed the appearance of the sweat, while in only five cases the leucocytes remained normal. The usual means of bringing about the perspiration was by the use of warm baths and the administration of hot tea or hot water, while from twelve to sixteen hours before the experiment was made no proteid food was given. The leucocytes in seven children were increased less than three thousand, in fifteen cases between three thousand and five thousand, while, as a rule, they commenced to return to normal within a half-hour after the child had been dried. The probable cause of the leuco-

¹ Centralbl. f. innere Med., 1900, No. 3.

² Deutsches Arch. f. klin. Med., vol. lxx., Nos. 1 and 2.

³ Centralbl. f. innere Med., Nov. 18, 1899.

⁴ Ibid., 1901, p. 823.

cytosis in these cases is the loss of fluid from the blood-vessels of the skin and the consequent concentration of the blood. Becker¹ concludes from his experiments upon the changes of the blood produced by vasomotor influence, especially cold baths and cold douches, that (1) the action of cold upon the entire body produces a slight increase of red corpuscles, (2) these changes are brought about partly by vasomotor influences, especially the abstraction of water from the blood and to a lesser extent by stasis of the blood-corpuscles in the capillaries, (3) the increase of leucocytes appears to be due in a great part to the formation of a peripheral zone, (4) in pathological conditions the changes of the blood are to be explained by an increased tendency towards stasis.

(b) THE INFLUENCE OF HIGH ALTITUDES.

Gottstein and Schroder² believe that the high blood counts in the case of individuals going from low to high altitudes are due entirely to the effects of the atmospheric pressure upon the blood-counting instrument, but Solly,³ who has studied the blood in twenty-five cases in Colorado, comes to different conclusions, for he has found that the red blood-corpuscles and hæmoglobin in visitors increase distinctly above that of the natives, while in the case of those who have been there for two or more years the blood count shows an intermediate position. Meissen and Schroder⁴ explain the more marked effect of altitude upon the red corpuscles and hæmoglobin in consumptives than in the case of healthy individuals by the chronic stasis in the circulation which is met with in tuberculosis of the lungs.

(VII.) DIFFERENTIATION OF HUMAN BLOOD FROM THAT OF ANIMALS.

From a medicolegal stand-point, the differentiation of human blood from that of animals is of the greatest importance, and yet it has only been within comparatively recent times that any satisfactory method for this differentiation has been devised. Thus before this method was devised, Däubler⁵ measured the red blood-cor-

¹ Deutsches Arch. f. klin. Med., vol. lxx., Nos. 1 and 2.

² Berl. klin. Wehnschr., July 2, 1900.

³ Phila. M. J., 1900, vol. i. p. 1074.

⁴ München. med. Wehnschr., vol. xlv., Nos. 2, 3, and 4.

⁵ Vrtljschr. f. gerichtl. Med., 1899, vol. xviii. 2, p. 258.

puscles of men and various animals in the hope of determining differences of sufficient magnitude to render differentiation possible, but he found that the differences were far too small to allow us to determine whether the blood is human or not. The recent work, however, on hæmolysis, seems to have definitely solved this problem. Uhlenhuth¹ has performed his experiments in this connection as follows:

Ten cubic centimetres of defibrinated human blood was injected into the peritoneal cavity of a rabbit at intervals of six days, and after five such injections an effective serum could be obtained. The blood of eighteen different animals was obtained and diluted with water, one to one hundred, and filtered. Of this clear, slightly red solution, two cubic centimetres were placed in a small tube and mixed with an equal quantity of 1.6 per cent. salt solution; six to eight drops of the serum of the rabbit were added to each of these tubes, but all remained perfectly clear except the tube containing human blood. The reaction is extremely delicate and can be obtained with very slight traces of blood, while it was also obtainable from blood which had been kept in dry condition for four weeks.

Deutsch,² Wassermann and Schultze,³ and Dieudonné⁴ describe practically the same method as Uhlenhuth and obtained the same results. The first of these claims to have been the first to have used this method of differentiating the human blood, while the last found that the same result could be obtained by human urine and human pleural exudate, although to a less degree.

(VIII.) THE ANÆMIAS.

No one subject in hæmatology has received more attention in the past few years than that of the anæmias. Broadly speaking, we may divide anæmias into primary and secondary, in the former of which there is no assignable cause for the blood condition outside of the changes in the hæmatopoietic apparatus, while in the latter the anæmia is secondary to some other pathological condition. Many observers hold that there are other underlying causes

¹ Deutsche med. Wehnschr., Feb. 7, 1901.

² Orvosik Lapja, 1901, No. 11.

³ Berl. klin. Wehnschr., 1901, vol. xxxviii., No. 7.

⁴ München. med. Wehnschr., 1901, No. 14.

of the so-called primary anæmias, but certainly, up to the present time, no one of these causes has been sufficient to explain the whole series of cases of that especial primary anæmia. In our consideration of the anæmias, after considering the question of anæmias in general to a slight extent, we shall take up in order primary pernicious anæmia, chlorosis, secondary anæmias, leukæmia, pseudoleukæmia, splenic anæmia, and finally we shall touch briefly upon the treatment of anæmia. The great difficulty in determining the exact nature of anæmia in certain cases is especially insisted upon by Lipowski.¹ He believes that severe anæmia, finally leading to the death of the patient, where no primary cause of the anæmia can be made out *intra vitam* or *post mortem*, must be considered a case of primary pernicious anæmia, even if the characteristic structures in the blood cannot be found, and thus differs from Ehrlich, who believes that the blood picture alone is sufficient to enable one to make the diagnosis of pernicious anæmia; he mentions several interesting cases in this connection.

Descatello and Hofbauer² discuss at length the subject of leucopenic anæmias. Besides the diminution of the leucocytes which occurs in typhoid fever, malaria, and measles, these investigators have found it also in five cases of pernicious anæmia, in four of chlorosis, in two of post-hemorrhagic anæmia, in two of cirrhosis of the liver, in one of phthisis floridans, in one of sepsis, in three of unknown origin, in two of pseudoleukæmia, and in two of splenic anæmia. They subdivide these cases into two groups, (1) in which the anæmia was not associated with glandular swelling, in which group the percentage of lymphocytes was constantly higher than normal, and (2) those in which disease of the glands was also present, in which group the percentage of lymphocytes was variable. They believe the leucopenia to be due to injury to the seats of blood formation, especially the marrow, although they regard it as an evidence of functional rather than of organic trouble, as Ehrlich holds.

Strauss and Rohnstein³ also pay special attention to the leucocytes in the various anæmias. From a study of fourteen cases of

¹ Deutsche med. Wehnschr., May 24, 1900.

² Ztschr. f. klin. Med., vol. xxxix. p. 488.

³ Berl., Hirschwald, 1901.

anæmia, they conclude that the leucocytes are decreased, as a rule, in pernicious anæmia and Banti's disease, and often, also, in other idiopathic anæmias, while in anæmias after carcinoma and sepsis they are regularly so, and often, also, in cases due to tuberculosis or cirrhosis of the liver and severe cases of chlorosis; in simple chlorosis they are normal. As to the number of lymphocytes in the various forms of anæmia, there was regularly a considerable increase in pernicious anæmia and Banti's disease, while in the other anæmias, especially that secondary to carcinoma, there was a relative decrease of the lymphocytes and a relative increase of the polymorphonuclear forms. This last observation is of especial interest in the differential diagnosis of pernicious anæmia and gastric carcinoma.

(a) PERNICIOUS ANÆMIA.

The term pernicious anæmia is used differently by different clinicians. Thus, one group regards pernicious anæmia as any anæmia which ends fatally, while the other group believes that pernicious anæmia is to be confined to those cases which are idiopathic or primary, that is, where no underlying cause of the anæmia is demonstrable outside of the hæmatopoietic apparatus. A number of investigators have attempted to show that those cases which, in the more strict of the above definitions, are to be considered as pernicious anæmia, are not really idiopathic, but are due to an auto-intoxication from the gastro-intestinal tract. Thus Stengel¹ believes the gastro-intestinal tract is the source of the hæmolytic agent, although he admits the possibility of the hæmogenetic function of the bone-marrow being perverted. He disagrees with Ehrlich in the latter's view that the association of megaloblasts with megalocytes is pathognomonic.

Hunter² and Elder³ also regard pernicious anæmia as a toxæmia, the former recommending as a logical treatment of this condition the use of antiseptics in the mouth and digestive tract; the latter reporting a case of pernicious anæmia cured with anti-streptococcus serum. Barker and Hunter⁴ report a fatal case of pernicious anæmia with characteristic blood picture consecutive to a traumatic stricture of the small intestine, although a considera-

¹ Med. News, Oct. 20, 1900.

² Lancet, Jan. 27, 1900.

³ Ibid., Apr. 28, 1900.

⁴ Ibid., July 21, 1900.

tion of their case makes one suspect that the case in reality might have been one of infection associated with severe anæmia. Shau-mann¹ believes that autointoxication is the best explanation of the etiology and pathogenesis of this disease, reaching his conclusions from the fact that in animals similar blood conditions are met with after the use of various poisons, from a study of the anæmias due to intestinal parasites, from the improvement in cases of anæmia secondary to various infections and intoxications when the gastro-intestinal disturbances are properly corrected, and from the increased proteid destruction and the characteristic changes in the spinal cord. While all these views are extremely interesting, nevertheless one cannot help feeling that in all these cases the gastro-intestinal disturbances could quite as well be regarded as secondary to the anæmia as the reverse; for, obviously, in any condition associated with such marked blood changes, secondary changes in all the organs and tissues are unavoidable. Thus Abrams² reports two cases in which the gastric symptoms were so marked as to make a diagnosis of gastric carcinoma seem probable, yet both had the characteristic blood picture of pernicious anæmia and improved immediately and progressively on arsenic. Also spinal-cord changes, consisting especially of degeneration of the posterior root columns, pyramidal and cerebellar tracts, as described by Von Voss,³ are best explained as due to the disturbances of metabolism resulting from the anæmia, and Strauss,⁴ after a most careful consideration of the relation between aepsia gastrica and pernicious anæmia, concludes that we possess absolutely no positive proof that an atrophy of the mucous membrane of the stomach and intestine can lead to pernicious anæmia. Cases with very acute courses are reported by Theodor⁵ and Muir.⁶ In the first of these the blood picture was typical; in the second there was an absence of megalyocytes and nucleated reds.

Thus we feel with Ehrlich that pernicious anæmia must for the present, at least, be regarded as idiopathic and characterized

¹ Samml. klin. Vortr., N. F., No. 287.

² Med. Rec., Apr. 28, 1900.

³ Deutsches Arch. f. klin. Med., vol. lviii. p. 489.

⁴ Ztschr. f. klin. Med., vol. xli. p. 280.

⁵ Wien. med. Wehnschr., No. lviii. p. 489.

⁶ Brit. M. J., Sept. 29, 1900.

in the vast majority of cases by a special blood picture, by a marked diminution of the red corpuscles, by a high color-index, often greater than one, by an absence of pallor in the red blood-corpuscles, and by the presence of megalocytes, poikilocytes, and nucleated red cells, while prognostically, the more marked the diminution of leucocytes and the greater the number of megaloblasts, the more serious is the outlook. To show the infrequency with which pernicious anæmia is met, Colman¹ has collected all the cases from six of the large general hospitals in Edinburgh. Of two hundred and fifty-two thousand six hundred and fifty-nine patients, but two hundred and forty-nine cases of this disease were met with, and of these thirty-seven were believed to have been cured, while one hundred and twenty-two died. Men were much more frequently affected than women, while as regards age, the youngest was seven, the oldest seventy-three, while the majority were between thirty-five and forty years. Stengel² believes that the diagnosis is assured in pernicious anæmia by an association of poikilocytosis of the highest grade, the presence of large numbers of megalocytes, marked polychromatophilia, and the presence of megaloblasts. As to the chemical constitution of the blood in pernicious anæmia, Rumpf³ finds that in this disease there is an increased amount of water, a decreased amount of dry residue, and a large amount of chlorides, but poverty in potassium, iron, and fat. He has attempted to make use of these findings therapeutically by administering potassium salts, especially the tartrate and citrate, in this disease, and reports three cases markedly improved under this treatment. Von Moraczewski,⁴ from his studies in metabolism in the severe anæmias, concludes that if the anæmia shows a marked chloride elimination and an absolute loss of calcium associated with a decreased nitrogenous excretion, the case is one, probably, of pernicious anæmia.

(b) CHLOROSIS.

The blood in chlorosis offers a distinctly different picture from that in pernicious anæmia, for here the red blood-corpuscles are diminished to a very much less extent than the hæmoglobin, that

¹ Edinb. M. J., Mar. and Apr., 1901.

² Med. News, Oct. 20, 1900.

³ Berl. klin. Wehnschr., 1901, No. 18.

⁴ Virchow's Arch., vol. clix., No. 2.

is, the color-index is extremely low, while the fresh and stained specimens show a marked pallor of the individual corpuscles with great tendency to microcytosis. This disease must still be regarded as idiopathic, notwithstanding the fact that various theories have been evolved to account for the condition. Thus Ischeroff¹ refers to the old theory of Clarke that the disease is due to autointoxication in the intestinal tract, while Hofmann² believes it to be due to an acquired or inherited hypoplasia of the bone-marrow. Grawitz,³ on the other hand, considers that the disease is rather part of a general neurosis than a primary blood disease, and, in its treatment, insists upon the removal of the cause of this neurosis, if possible, by diet, change of air, and often by removal and isolation of the patient, by the use of wet packs, etc.

Jolasse⁴ insists that besides rest and over-feeding, iron is necessary, and recommends suppositories of iron citrate, while Terrile and Curlo⁵ recommend the subcutaneous administration of iron. Hofmann,⁶ from experiments performed upon rabbits, to determine the rôle of iron in blood regeneration, concludes that the iron stimulates the physiological activity of the bone-marrow and brings about a more rapid ripening of the erythrocytes, but does not directly influence the formation of hæmoglobin, while he believes that chlorosis, in all probability, consists of either a more or less marked hypoplasia of the bone-marrow inherited and manifesting itself during the whole life of the individual, or a diminished activity of the bone-marrow manifesting itself only at the time of puberty.

The former of these two conditions in severe cases may be associated with hypoplasia of the heart and blood-vessels, or even of the genital apparatus, and is described by Virchow. This weakness of the blood-forming organs expresses itself in the production of red blood-corpuscles irregular in form and deficient in size and hæmoglobin.

Leclerc Levet,⁷ from a consideration of the family history of

¹ *Jahrb. f. Kinderh.*, vol. xlv., No. 4, p. 393, and vol. xlv., Nos. 1 and 2, p. 153.

² *München. med. Wehnschr.*, 1899, No. 29.

³ *Therapie der Gegenwart*, June, 1900.

⁴ *München. med. Wehnschr.*, 1899, No. 37.

⁵ *Clin. med. Ital.*, 1899, No. 10.

⁶ *Virchow's Arch.*, vol. clx. p. 2.

⁷ *Lyon Méd.*, 1901, No. 31.

chlorotics, concludes that neuropathic, gouty, and tuberculous conditions are frequently met with, while his experiments upon the blood-serum in chlorosis seem to show that it is much more toxic than normal serum.

(c) SECONDARY ANÆMIAS.

The subject of secondary anæmia will, of course, be considered in detail in our consideration of the changes in the blood in various diseases and pathological conditions. Here we shall simply discuss the general subject of secondary anæmia, especially in relation to some of the work recently done in this connection. The varieties of secondary anæmia are many; thus we may have oligochromæmia, or oligocythæmia, or, as is usually the case in secondary anæmia, a combination of these two, or oligæmia, in which the total quantity of the blood in the body is diminished, so that, although the determination of hæmoglobin and of red corpuscles gives a normal count, the clinical symptoms of anæmia, shortness of breath, pallor, hæmic murmurs, etc., are present. In the great majority of cases of secondary anæmia, the hæmoglobin is reduced to a distinctly greater extent than the red corpuscles; thus we have a low color-index and, on microscopic examination, distinct pallor of the red blood-corpuscles, although the difference between the degree of reduction of hæmoglobin and of red corpuscles is usually less than in the case of chlorosis. The secondary or symptomatic anæmias may be, broadly speaking, divided into three classes, those due to loss of blood; those due to poor nourishment, unhealthy mode of life, and bad hygienic conditions; and those due to various organic or functional diseases, or diseases due to parasites, or after the administration of various poisons. In all these conditions the degree of the anæmia depends upon the severity and extent of the cause, and, in the very severe cases, we may have as marked changes in size and shape of the red blood-corpuscles as in cases of pernicious anæmia, while nucleated red blood-corpuscles may also be met with.

Ewart¹ describes a case of puzzling anæmia which was finally found to be due to completely ignored hemorrhoidal nodules high up in the rectum, and the author of this review has met with a similar case which had previously been diagnosed by some as per-

¹ *Therapie der Gegenwart*, Nov., 1899.

nicious anæmia, by others as carcinoma. Such cases as these should make us extremely careful in diagnosing primary anæmia, and every portion of the body should be most carefully examined in the hope of discovering a primary cause before such a diagnosis is made.

Senator¹ suggests that, as malignant growth of the bone-marrow is sometimes the cause of anæmia, in all doubtful cases the urine should be examined for albumose, as albumosuria is significant of malignant disease of the bones.

Rosenquist² has performed a series of experiments to determine whether experimental anæmia is associated with an increased proteid destruction, as first insisted upon by Von Noorden. These studies were carried on in cases of anæmia produced by the bothriocephalus, and in all fifteen cases there was a reduction of nitrogen. Rosenquist thinks that this is not entirely at the cost of the blood, but that the other tissues also take a part in this increased breaking down of proteid material. As a rule, the destruction of nitrogenous material runs parallel with the diminution of the red blood-corpuscles.

Kaminer and Rothstein³ have used phenylhydrazin in producing experimental anæmia in animals. After doses large enough to kill in forty-eight hours, the red blood-corpuscles were reduced to one million or lower; macrocytes, fragmented and polychromatophilic erythrocytes were present, as well as normoblasts and a few megaloblasts, while there was also a moderate leukopenia. By using a smaller dose, a chronic anæmia could be obtained with reduction of red cells, presence of macrocytes, and a few nucleated red cells, but no poikilocytes; usually there was a leucocytosis.

(d) LEUKÆMIA.

In leukæmia, blood examination is of paramount importance, for by its means alone are we able to differentiate the lymphatic type of this disease from Hodgkin's disease, syphilitic or tuberculous adenitis or sarcoma of the lymph-glands; or the splenomyelogenous type of this disease from Banti's disease, malignant growths of the

¹ Berl. klin. Wchnschr., July 23, 1900.

² Berl. Ver. f. innere Med., Centralbl. f. innere Med., 1901, p. 521.

³ Berl. klin. Wchnschr., July 30, 1900.

spleen, enlarged spleen due to malaria or syphilis, or large renal or ovarian neoplasm, occupying the entire left half of the abdominal cavity. As to the blood picture in the two types of the disease, as is well known, it is absolutely different, for in lymphatic leukæmia we have an enormous increase of leucocytes, often reaching 100,000 to 200,000, and of these the greater proportion, often from ninety per cent. to ninety-eight per cent., are mononuclear non-granular forms, usually small lymphocytes. In the splenomyelogenous forms of the disease, on the other hand, the leucocytic increase is even greater, often reaching 400,000 to 500,000 per cubic millimetre, while the blood is characterized by the presence of large numbers of myelocytes, often thirty to sixty per cent., eosinophilic myelocytes and mast-cells, while there is also an enormous increase of the polymorphonuclear neutrophiles, although relatively they are decreased; in both cases marked anæmia is present, while in the case of splenomyelogenous leukæmia, as a rule, large numbers of nucleated corpuscles are to be found in the circulating blood.

As to the origin of the cells in this disease, although Ehrlich believes that in lymphatic leukæmia the cells that are markedly increased, the lymphocytes, are derived from lymph-glands, nevertheless, many recent investigators have concluded that the bone-marrow is the seat of formation of these cells; of course, practically all observers agree that the bone-marrow is the seat of formation of the white blood-cells in splenomyelogenous leukæmia. Thus Pappenheim,¹ from a careful experimental study upon animals and human beings, and from a consideration of all his cases of this disease, concludes that leukæmia arises only in case the bone-marrow yields to some pathological stimulus. Thus, if the red-marrow parenchyma is unchanged, we get myelogenous leukæmia; if the marrow is substituted by lymphoid tissue, lymphæmia or lymphatic leukæmia; while if the faulty stimulant affects only the spleen and lymph-glands, where the capsule is capable of being stretched, and thus growing with the gland, no cell-elements enter the circulating blood, and there arises pseudoleukæmia.

Walz² comes to the same conclusion as Neumann and Pappenheim, that is, that lymphoid hyperplasia of the bone-marrow leads

¹ Ztschr. f. klin. Med., vol. xxxix. p. 171.

² Württemb. Corr.-Bl., 1899, vol. lxix., No. 44.

to lymphatic leukæmia, while the pyoid or myelocytic hyperplasia leads to myelogenous leukæmia.

As to the etiology of leukæmia, the most interesting work has been that of Löwit in regard to his so-called parasites of leukæmia, although the great majority of observers absolutely oppose his views.

Löwit¹ describes in detail this supposed sporozoon, which he says was found by him in eleven cases of mixed leukæmia, three of lymphæmia (lymphatic leukæmia), and two cases of leukæmia only examined *post mortem*. As the number of his so-called sporozoa was greater in the leucocytes of the spleen than in those of the peripheral circulation, he thought that the spleen and probably also the other blood-forming organs were the places wherein the parasites developed. In the mixed leukæmia the parasites were found in the spleen, bone-marrow, and lymph-glands, while in the lymphatic leukæmia, though not present in the circulating blood, they were found in the hæmatopoietic organs. He therefore describes two varieties of parasite, one of which is found both inter- and intracellular in the blood-forming organs and in the circulating blood, and is the one seen in mixed (splenomyelogenous) leukæmia; and the other of which is found only in the blood-forming organs, is always intercellular, and is the one seen in pure lymphæmia (lymphatic leukæmia). So far all attempts to cultivate the protozoon have proven unavailing.

Türk² believes that the supposed protozoon of leukæmia described by Löwit is nothing more than an artefact composed of "Mastzellen" granulations, and he claims to have obtained them in normal rabbit's blood, in normal human blood, and in the blood in lues, anchylostomiasis, carcinosis, and other diseases, by Löwit's method of staining, but by no other method.

Vittadine,³ from his work at the Pathological Institute at Pavia, claims to have found Löwit's hæmamœba in three cases of leukæmia, while in a later communication Löwit⁴ gives the result of his further investigations, claiming that he has found his parasite in all cases of leukæmia, and that he could produce leukæmia experi-

¹ Wien. klin. Wchnschr., No. 20, 1898.

² Ibid., Mar. 29, 1900.

³ Gazz. d. Osp., 1900, No. 63.

⁴ Prag. Ztschr. f. Heilk., 1900, No. 10.

mentally in rabbits by the injection of blood from patients suffering from this disease. On the other hand, Türk,¹ in association with Decastello, has performed the same experiments as Löwit in regard to the inoculation of rabbits with leukæmic blood and obtained absolutely negative results.

Hirschfeld and Tobias² performed a similar series of experiments, also with negative results, and certainly at the present time the weight of proof is distinctly against the protozoon origin of leukæmia.

The effect of intercurrent infectious diseases upon leukæmia is a subject of really great interest, as it has been noted that during the course of various infections, typhoid, influenza, erysipelas, septic infections, etc., there is often a marked diminution in the size of the splenic tumor and in the number of leucocytes per cubic millimetre in the circulating blood.

Kormoczi,³ from the cases he reports and from a survey of the literature, concludes that this diminution is explained by the destructive action of the bacterial toxins upon the tissues, while the qualitative changes in the blood depend upon the chemotactic properties of these toxins. In different cases these two factors differ, so that in some the destructive and in others the chemotactic action of the toxins is more marked. In the latter case, the total number of white blood-cells may remain unchanged, while the relative proportions of the different varieties may change markedly; that is, the proportion of myelocytes decreases and the proportion of neutrophils increases.

Kraus⁴ reports an interesting case of splenomyelogenous leukæmia where the splenic tumor and the increase of the white blood-cells (360,000 per cubic millimetre) entirely disappeared during an intercurrent general infection following an attack of erysipelas.

Adler⁵ met with a rapid fall of leucocytes in two of his twenty-three cases of leukæmia, one without apparent cause, the other during an intercurrent septic infection. In the second case, the most noteworthy phenomenon was the enormous decrease of the

¹Wien. klin. Wchnschr., 1901, No. 18.

²Deutsche med. Wchnschr., 1902, No. 6.

³Ibid., 1899, No. 47.

⁴Prag. med. Wchnschr., 1899, Nos. 41 and 42.

⁵Ztschr. f. Heilk., 1902, Nos. 8 and 9.

myelocytes and eosinophilic myelocytes with relative increase of the lymphocytes.

A number of interesting and atypical cases of leukæmia have been recently reported, of which we shall mention but a few.

Gilbert and Weil¹ report three cases of acute leukæmia, besides collecting sixteen cases from the literature. In these three cases death occurred in seven weeks, one month, and fifteen days, respectively.

McCrea² reports a case of acute lymphatic leukæmia where death occurred in four weeks, and has collected thirteen other cases of acute leukæmia in children from the literature.

Arneth³ reports a case of rapidly fatal anæmia in a boy ten years of age, the course of the disease lasting only three weeks. The red blood-corpuscles were reduced to 250,000 per cubic centimetre, while the leucocytes were especially interesting, for, although not absolutely increased, the relative proportion of the various forms was markedly changed; the lymphocytes were much increased, many of the polymorphonuclear cells possessed other granulations besides the neutrophilic, and myelocytes of all sizes were met with. The examination of the red bone-marrow showed all the various forms of the red and white cells described above and also peculiar cells which Arneth believes to be transitional forms between myelocytes and nucleated red blood-corpuscles. The spleen was much enlarged and contained large numbers of lymphocytes, but the lymph-glands were normal.

Samman⁴ reports a case of lymphatic leukæmia in which death occurred eleven days after the appearance of the onset of the symptoms. An atypical case of myelogenous leukæmia is reported by Hirschfeld and Alexander,⁵ the fact of especial interest in connection with this case being the complete absence of the eosinophiles and mast-cells from the circulating blood, although the post-mortem examination showed it to be a case of myelogenous or possibly combined leukæmia. Hirschfeld and Alexander believe that this ab-

¹ Arch. de méd. expér., 1899, p. 157.

² Johns Hopkins Bull., May, 1899.

³ Deutsches Arch. f. klin. Med., vol. lxi., Nos. 3 and 4.

⁴ Brit. M. J., Feb. 23, 1901.

⁵ Berl. klin. Wchnschr., 1902, No. 11.

sence was due to the great trauma to the bone-marrow as evidenced by the extremely rapid course of the disease.

Von Jaksch¹ reports a case of multiple periosteal disease associated with blood conditions suggestive of myelogenous leukæmia in a girl, who suffered with severe pains in the bones and joints, and in whom examination showed a marked enlargement of the spleen and 40,000 to 50,000 leucocytes per cubic millimetre, among which were numbers of myelocytes; there were also numerous nucleated red corpuscles. The X-ray showed marked thickening of the periosteum of the affected bones, while the blood picture, as the case progressed, showed a diminution of the white corpuscles. Von Jaksch concludes that the case is either an atypical form of myelogenous leukæmia, or possibly a disease of the bone due to a periosteal neoplasm.

Rosenfeld² describes three cases of lymphatic leukæmia, (1) lymphosarcomatosis of various organs with large numbers of lymphocytes in the blood, (2) enlargement of the lymph-glands and a diffuse lymphadenoid degeneration of the bone-marrow, while the blood showed large as well as small mononuclear lymphocytes, (3) a typical case of the disease.

From these cases Rosenfeld concludes that swelling of the lymph-glands *per se* does not lead to any considerable increase of lymphocytes, but when the bone-marrow is affected (lymphadenoid degeneration), the blood picture changes, the red blood-corpuscles decrease, and besides the small lymphocytes large forms make their appearance. Associated with this change in the blood picture, the clinical symptoms frequently become more severe, and the disease rapidly reaches a fatal termination. As to the prognosis in myelogenous leukæmia, Taylor describes marked degenerative changes in the leucocytes towards the end of the disease.

(e) PSEUDOLEUKÆMIA. (f) SPLENIC ANÆMIA.

Most authors regard splenic anæmia or Banti's disease, and lymphatic anæmia or Hodgkin's disease, as the splenic and lymphatic types respectively of the same disease, pseudoleukæmia. Banti, however, insists that splenic anæmia or splenomegaly should

¹ Prag. med. Wehnschr., 1901, Nos. 1 and 2.

² Ztschr. f. klin. Med., vol. xlii. p. 117.

not be considered as the splenic type of pseudoleukæmia but as a separate disease, characterized by four distinct stages, enlargement of the spleen, anæmia, transition, and ascites.

Osler¹ reports fifteen cases of splenic anæmia, twelve in men, three in women, in all of which the splenic enlargement seemed to precede the anæmia.

In all cases of pseudoleukæmia the leucocytes show practically no changes, which is especially interesting in connection with Pappenheim's and Rosenfeld's views regarding the etiology of the two types of leukæmia, that is, that the blood-changes do not appear because of the changes in the spleen in the one case, or of the lymph-glands in the other, but not until myelocytic or lymphoid hyperplasia is present in the bone-marrow.

Türk² makes a plea for a more scientific nomenclature for the various pseudoleukæmias, suggesting "lymphomatosis partialis," "lymphomatosis universalis," "lymphomatosis destruens," and "lymphosarcomatosis," according to the pathological process present in the individual case.

The anæmia infantum pseudoleukæmica, as originally described by Von Jaksch and recently by Stein,³ in which a condition is met with characterized by anæmia, enlargement of the spleen, swelling of the liver, and much emaciation, and where the blood usually shows an enormous number of normoblasts and megaloblasts, and a quite marked leucocytosis, is regarded at the present time as probably not a primary anæmia but an anæmia secondary to rickets.

(g) THE TREATMENT OF ANÆMIA.

As to the treatment of anæmia, much has already been said under the special headings, and we shall here simply insist upon the necessity of paying attention to the dietetic and hygienic treatment of the cases, as well as to the medicinal. In many cases of anæmia of a severe type, rest, sunshine, careful feeding, and fresh air, are equally as important as the administration of iron or of arsenic. As to the medicinal treatment of the disease, in all of the secondary anæmias and in chlorosis, iron is indicated, while arsenic is also indicated in all cases of primary pernicious anæmia

¹ Am. J. Med. Sc., Jan., 1900.

² Wiener klin. Wchnschr., 1899, No. 40.

³ Therap. Monatsch., 1899, No. 10.

and leukæmia, besides being frequently of value in many of the secondary anæmias. The great trend of modern scientific opinion is against the so-called organic iron preparations and hæmoglobin preparations. Thus Marcuse,¹ experimenting with a large number of these proprietary remedies, concludes that the use of all these preparations is distinctly irrational, because in whatever form administered, the iron is changed in the stomach and duodenum before being absorbed, and thus the use of inorganic preparations is much more rational, because they contain a larger proportion of iron and are just as easily changed and absorbed.

According to Aporti,² the production of hæmoglobin is increased by the injection of iron, and the production of red blood-cells by the injection of arsenic, although this view is not universally held.

Aporti and Camillo³ found that of all heavy metals, iron and manganese alone produced an increase of red corpuscles and hæmoglobin.

The injection of normal salt-solution, in post-hemorrhagic anæmia, has been much used of late, and Taylor⁴ has recently contributed an interesting article upon the restitution of the blood-plasma in this condition.

(IX.) LEUCOCYTOSIS.

In discussing leucocytosis, it is essential to first draw the distinction between physiological leucocytosis, that is, leucocytosis met with under normal physiological conditions, and pathological leucocytosis where some disease or pathological condition is the underlying cause.

(a) PHYSIOLOGICAL LEUCOCYTOSIS.

Physiological leucocytosis is met with during the digestion of foodstuffs, in pregnancy and in the puerperium, in infancy, and after violent exercise, massage, electricity, and cold baths, while the so-called terminal leucocytosis described by many as occurring just before death, is believed by some to be due to blood-stasis, by others to be due to a general terminal infection. The digestion leucocytosis, usually appearing from one to two hours after a meal, espe-

¹ Arch. d. Heilk., 1901, Nos. 4 and 5.

² Centralbl. f. innere Med., Jan. 13, 1900.

³ Clin. med. Ital., 1900, No. 8.

⁴ Contributions from the Pepper Laboratory, Phila., 1900.

cially if much proteid food has been taken, is of interest, because of the fact that it is absent under certain conditions, such as debility, disturbances of the gastro-intestinal tract, and in most cases of carcinoma of the stomach. In this last connection, the absence of the digestion leucocytosis is often a real help to us in making the diagnosis of malignant disease of the stomach.

The increase is usually from twenty-five per cent. to thirty-five per cent., while according to most observers, the relative proportion of the various leucocytes remains constant, although some describe a relative increase of the neutrophiles, others of the lymphocytes.

The leucocytosis of pregnancy is usually met with in the case of primiparæ, less frequently in the multiparæ, while most observers have met with a distinct leucocytosis *post partum*.

The leucocytosis of infancy has already been described, while the leucocytosis after exercise, massage, cold baths, and electricity is probably due to the stimulation of the vasoconstrictors and the corresponding concentration of the blood. Carstanza¹ has carried on a careful series of experiments regarding the digestion leucocytosis. He found that the number of polymorphonuclear leucocytes is generally higher before meals than a few minutes after; sometimes they are increased for a short time after a meal, but, as a rule, their minimum is reached three to four hours after a meal, from which time they again increase. The relative number of the lymphocytes before and after the meals he found to be the reverse of that of the polymorphonuclear leucocytes, while the transitional forms and the eosinophiles are not influenced by digestion.

A practical point in connection with physiological leucocytosis is that the blood should never be examined for pathological changes during the height of digestion, after violent exercise, cold baths, or massage, but rather on a fasting stomach, when the patient is at rest, so that the degree of leucocytosis may be a criterion of one factor alone, and not a combination of two factors, one physiological and one pathological.

(b) PATHOLOGICAL LEUCOCYTOSIS.

The subject of pathological leucocytosis will, of course, be most carefully considered under Section XI., where the changes in the

¹ Jahrb. f. Kinderh., vol. lii., Nos. 2 to 4.

blood in the various diseases and pathological conditions are taken up, so that here we shall simply discuss briefly the significance of the various types of pathological leucocytosis, the polymorphonuclear neutrophilic type, the polymorphonuclear eosinophilic type, and the lymphocytic type. In discussing leucocytosis, we should remember that it usually represents the conflict of two factors, virulence of infection the one, resistance of the patient the other. Cabot expresses this well in the following formula:

“With a mild infection and a good vital reaction, there is a small leucocytosis; if the infection is less mild, vital reaction less good, there is a moderate leucocytosis; if the infection is severe but the vital reaction good, there is a very marked leucocytosis, while if the infection is severe and the vital reaction poor, there is no leucocytosis and often a leucopenia.” This last point is of extreme importance, because if in pathological conditions normally associated with leucocytosis, no leucocytic increase is met with, the prognosis is very grave, in fact, usually fatal, especially if the leucopenia is marked.

(1) *Polymorphonuclear Neutrophilic Leucocytosis*.—The polymorphonuclear neutrophilic leucocytosis, or that type characterized by an increase largely confined to the polymorphonuclear neutrophils, is the type usually met with. This condition is met with in a great number of pathological conditions, among which we may mention most of the inflammatory and infectious diseases, a large number of intoxications, after the use of many drugs, after severe hemorrhages, and in malignant diseases, whether carcinoma or sarcoma. The increase of leucocytes in these conditions is supposed to be due to the presence of substances which have a positive chemotactic effect upon this form of leucocyte. Nakanishi¹ has shown the resistance of the leucocytes to destructive influences, and has found by means of his method of vital staining that they are able to preserve their life from ten to twenty-eight days after removal from the body. Some observers, notably Horbaczewski, have attempted to show that leucocytosis is invariably associated with an increase in the uric-acid excretion, but Douglas² and others have shown that there was no true correlation between the leucocyte count

¹ München. med. Wehnschr., May 1, 1900.

² Edinb. M. J., 1900, No. 1.

and the uric-acid excretion, while T. R. Brown¹ has demonstrated the same absence of correlation between eosinophiles and uric-acid excretion.

(2) *Polymorphonuclear Eosinophilic Leucocytosis.* — As we have said before, the eosinophiles normally present constitute one to two per cent. of all the leucocytes, but an increase of these cells, that is, eosinophilia, is met with in a number of conditions. As to the exact normal percentage of these cells, there is still some difference of opinion, but the majority of careful observers give between one and four per cent., while Zappert expresses their quantity better, perhaps, by giving the number present per cubic millimetre, regarding fifty to one hundred as low normal, one hundred to two hundred as intermediate, two hundred to three hundred as high normal, and over three hundred as pathological. According to Zappert, over four per cent. must be regarded as pathological if the total number of leucocytes is not increased, while, according to Wolff,² over six to eight per cent. is regarded as pathological; of course, if the leucocytes are numerically increased per cubic millimetre, a smaller percentage of these cells will show that they are increased.

Klein³ believes the presence of eosinophile-cells in diseased areas is not pathognomonic of some specific disease, but is brought about by the extravasation of a certain amount of blood into the tissues which is absorbed by the neutrophiles; but this view is highly improbable, as especially insisted upon by Piotrowski and Zaleski,⁴ who insist that if this were the case, an eosinophilia should be met with in all cases associated with extravasation of blood, as fractures, contusions, infarcts, pneumonia, etc., and should not be present in conditions not associated with hemorrhage, as bronchial asthma.

Hankin has always insisted that the eosinophile-granules play an important rôle in the formation of alexins, while Noesske⁵ concludes that the eosinophiles play the important rôle of protectors against the invasion of bacteria.

¹ J. Exper. Med., vol. iii., No. 3.

² Ziegler's Beiträge, vol. xxviii., No. 1.

³ Centralbl. f. innere Med., 1899, Nos. 4 and 5.

⁴ Ibid., 1899, No. 22.

⁵ Deutsche Ztschr. f. Chir., Apr., 1900.

According to Ehrlich, the substances most positively chemotactic to eosinophiles are those due to the breaking down of epithelial cells. Eosinophilia is met with in a number of conditions, as bronchial asthma, pemphigus, various acute and chronic skin-diseases, various diseases due to intestinal parasites, trichinosis, frequently after the termination of a fever, sometimes in malignant tumors, after the extirpation of the spleen, and after the administration of various drugs and poisons, while a moderate eosinophilia is occasionally met with in a great variety of other conditions.

(c) LYMPHOCYTOSIS.

Lymphocytosis, the passive leucocytosis of Ehrlich, or that condition in which we have a relative increase of lymphocytes, usually at the expense of the neutrophiles, is met with under a variety of conditions in infancy and early childhood, and in a few of the infectious diseases, notably, whooping-cough, while Türk and Saquepée have described it in mumps, Courmont, Montagard, and Weil in smallpox and varicella, and a relative increase of the lymphocytes has been described in typhoid fever, malaria, and in cases of anæmia of great gravity and grave prognosis, although in these last conditions the total number of leucocytes is not increased.

(X.) LEUCOPENIA.

Leucopenia or hypoleucocytosis is met with in a number of conditions, and is as useful in aiding us to make a differential diagnosis as the opposite condition. Whether it is due to an unequal distribution of the leucocytes (a diminution in the peripheral and heaping up in the central circulation), or whether it is due to the development of negatively chemotactic substances, has not been as yet definitely determined; probably each of these factors is true in a certain number of cases. Leucopenia is met with in a variety of conditions, after short hot baths, or prolonged cold baths, in conditions associated with lack of food, usually in pernicious anæmia, occasionally in other anæmias, especially if they lead to a fatal termination, in various inflammatory and infectious conditions where the reaction of the patient is very poor and the prognosis very grave, and in the course of a number of infectious diseases, as uncomplicated typhoid fever, malaria, measles, influenza, especially the abdominal type of the disease, and uncomplicated tuberculosis.

In this leucocytic decrease, the neutrophiles, as a rule, are the cells most affected.

(XI.) THE BLOOD IN SPECIAL DISEASES AND PATHOLOGICAL CONDITIONS.

(a) THE BLOOD IN INFECTIOUS DISEASES.

(1) *Tuberculosis*.—The study of the blood in tuberculosis is of great value for many reasons. The great majority of observers have found an absence of leucocytosis in uncomplicated cases of tuberculosis, while a number report a distinct diminution of leucocytes in this disease, especially in the intestinal and peritoneal types. In this latter condition there is frequently a relative increase of the mononuclears, and according to Achard and Loeper, this latter condition is met with in the case of tuberculosis of any of the serous membranes. Dembinski¹ has experimented in this connection by inoculating guinea-pigs with the tubercle-bacillus. He found that a leucocytosis resulted, polymorphonuclear for the first two days, and mononuclear from the third day on. The prognostic value of eosinophiles in the blood and sputum of tuberculosis has been insisted upon by a number of observers, although there are not wanting other investigators who oppose these views. According to the former, notably Teichmüller, the presence of the eosinophiles in the sputum is favorable prognostically.

Memmi,² from the examination of the sputum in thirty-five cases of tuberculosis, found that no eosinophiles were found in cases of beginning tuberculosis, that, as a rule, such leucocytes were more frequent in patients who showed a marked improvement, although they were sometimes met with in cases of advanced tuberculosis, while after a hæmoptysis the cells were markedly increased.

A consideration of all the results in this connection seems to show that too much reliance must not be put on this sign in the prognosis of tuberculosis. The absence of leucocytosis in uncomplicated tuberculosis is of value, in the first place, in differentiating tuberculous from other inflammations of various organs and tissues characterized by an identical train of symptoms, while the appearance of a leucocytosis in tuberculous cases points at once to there

¹ Gaz. hebd. de méd., Jan. 11, 1900.

² Gazz. d. osp., 1901, No. 114.

being a secondary infection with pyogenic micro-organisms, which may be of the greatest value in determining upon immediate operation, in the bone- and joint-cases especially. Secondary anæmia, so often met with in tuberculosis, may be due to the infection with the tubercle bacillus, but is more likely to be the result of a secondary infection.

(2) *Pertussis*.—The blood in whooping-cough has been especially studied by Frölich and Meunier, and by De Amicis and Pacchioni.¹ These investigators found that a leucocytosis is regularly present in this disease and is more marked than in any other disease of the respiratory tract except, perhaps, lobar pneumonia. The latter found the average number of leucocytes in this disease to be 17,943, the leucocytosis beginning on the first day of the disease, reaching its height in the convulsive stage, and still demonstrable some time after the cessation of the typical symptoms. The increase seems to be especially confined to the mononuclear elements, the small mononuclears being especially numerous in the first and second stages of the disease, the large mononuclears in the third stage. According to counts of others, the average number of leucocytes per cubic millimetre is 28,000, while in some cases the leucocytes reach 50,000 and above; the increase occurs early, during the catarrhal period, and is especially marked in very young infants.

Cima² has obtained the same results and believes that the leucocytosis is more marked, the more severe the case the smaller the child, and the more complications present.

(3) *Diphtheria*.—Practically all cases of diphtheria show leucocytosis except the mild and the most severe; in the latter of which the presence of leucocytes is a most unfavorable sign. Engel³ believes that the condition of the blood is a most valuable prognostic sign in this disease. From a study of thirty-two cases, he concludes that a high percentage of myelocytes is always unfavorable, although an increase of these cells is not met with in all the fatal cases.

Besredka,⁴ from a careful blood examination in animals with experimental diphtheria, and in forty-nine children with this dis-

¹ Clin. med. Ital., 1899, No. 1.

² *Pediatrics*, 1899, No. 9.

³ *Deutsche med. Wchnschr.*, 1897, vol. xxiii., No. 8.

⁴ *Ann. de l'Inst. Pasteur*, 1898, vol. xii. 5, p. 305.

case, concludes that the grade of the polymorphonuclear neutrophilic leucocytosis, after the administration of the antitoxic serum, gives the prognosis. Thus, if one to two days after the injection the percentage of these cells is sixty or above, the prognosis is good; with a higher temperature and fifty per cent., the prognosis is bad, while if below fifty per cent. the prognosis is fatal.

(4) *Typhoid Fever*.—The anæmia secondary to typhoid fever is interesting in that it does not make its appearance until quite late in the disease, but lasts for a long period into convalescence. According to Hayem, in this disease the leucocytes frequently fall from one thousand to two thousand per cubic millimetre, while Ohantemesse and Millet describe a slight increase during the first week, decreasing subsequently and persisting for a long time during convalescence. According to these latter observers, there is at first a slight relative increase of the neutrophiles and decrease of the lymphocytes, and complete absence of the eosinophiles, while later the formula is reversed.

Naegeli,¹ from an enormous number of investigations made upon fifty cases of typhoid fever, finds that the leucocytes undergo characteristic changes in the various stages of this disease. He thinks that these changes are due to the action of the typhoid toxins upon the bone-marrow, hindering the production of the neutrophiles and eosinophiles, while it is probable that the functions of the lymphatic tissues are also disturbed. -

The changes in the first stage of the disease (steadily rising temperature) are: a neutrophilic leucocytosis of moderate degree, rapidly decreasing until the neutrophiles are diminished, a disappearance of the eosinophiles, and a moderate decrease of the lymphocytes. In the second stage (continued fever) the polymorphonuclear neutrophiles and lymphocytes are still further decreased, although towards the end of this stage the latter tend to increase again. In the third stage (remission) the neutrophiles become fewer, the lymphocytes continue to increase, and a few eosinophiles appear. In the fourth stage (defervescence) the neutrophiles reach their minimum, the lymphocytes are greatly increased, and the eosinophiles gradually return to their normal number. As soon as the fever disappears the neutrophiles begin to increase again, but

¹ Deutsches Arch. f. klin. Med., vol. lxxvii., Nos. 3 and 4.

there is often, for some time, a considerable lymphocytosis. All these changes were more pronounced in children. According to Naegeli, favorable indications are the early appearance of the eosinophiles, the moderate diminution of the polymorphonuclear neutrophiles, and the extreme increase of the lymphocytes. In most complications, as perforation, etc., there is a leucocytosis of the neutrophilic type, although Kölner¹ finds that in his series of cases, if complicated with pneumonia or otitis media, the leucocytes were sometimes decreased, sometimes increased. It will be interesting to know, in the case of these last-mentioned complications, when no increase of leucocytes occurred, whether the complication was due to the typhoid bacillus and not to pyogenic micro-organisms.

A very interesting article in this connection has been contributed by Bohland² on the chemotactic action of the toxins of the typhoid bacillus and the colon bacillus on the leucocytes; he found from animal experiments that the toxins of the typhoid bacillus were regularly negatively chemotactic, while the reverse was true in the case of the colon bacillus.

Houston³ opposes the views of the majority of observers that the decrease of the polymorphonuclear neutrophiles in typhoid fever depends upon negative chemotaxis, bringing forward as arguments that the polymorphonuclear decrease bears no relation to the intensity of the toxæmia, and that moribund cases are frequently met with where neither neutrophiles nor hæmoglobin are markedly diminished. He believes that the diminution of neutrophiles is much better explained by the dilution of the blood which, he thinks in turn, depends upon the increased activity of the lymphatic apparatus, although in many cases the diarrhoea and sweating, by their concentrating effects, compensate for this dilution.

The great value of leucocyte counting in typhoid fever is in differentiating this disease from central pneumonia, appendicitis, various inflammatory and suppurative processes, such as suppurative ovarian cysts and pyosalpinx; in the second place, after the diagnosis of typhoid fever has been established, in giving us a most

¹ Deutsches Archiv. f. klin. Med., 1898, vol. lx., p. 221.

² Centralbl. f. innere Med., 1899, No. 17.

³ Brit. M. J., Feb. 15, 1901.

valuable symptom that some complication, as intestinal perforation, has taken place, and in the asthenic cases, in which symptoms are slight, due to the intense toxæmia, this may be the only means of diagnosing such conditions and, *ipso facto*, operating and possibly saving the patient's life.

(5) *Scarlet Fever*, (6) *Measles*.—Almost all observers, as Van der Berg,¹ etc., describe a leucocytosis lasting from twenty to thirty days in scarlet fever. Kotschetkoff was the first to call attention to the fact that during the first and second weeks of scarlet fever, in favorable cases, there was a distinct increase of the eosinophiles, sometimes reaching fifteen per cent., while in the fatal cases this increase was not met with; these observations have been substantiated by Zappert, T. R. Brown, and others. The average number of leucocytes in these cases per cubic millimetre lies between 10,000 and 30,000; thus blood counting is of value in diagnosing this disease from measles.

In measles, the leucocytes are either normal or sub-normal, although Renaud describes a slight leucocytosis during the period of incubation of the disease. In this disease there is also sometimes a slight relative increase of the lymphocytes. A valuable article upon the blood in measles and scarlet fever in children is that of Reckzeh² in which he states that he has found leucopenia with relative increase of the lymphocytes in measles, and leucocytosis with relative increase of the neutrophiles, and later also of the eosinophiles in scarlet fever.

German measles is also not associated with an increase of leucocytes.

(7) *Syphilis*.—Monod³ lays especial stress upon the prevalence of anæmia in syphilitics. He thinks it is one of the earliest manifestations of the infection, preceding the roseola and associated with a leucocytosis which is generally the last of the blood changes to disappear. According to Monod, there is an absolute concordance between the anæmia on the one hand and the appearance of the specific symptoms on the other, as well as a direct relation between the grade of the anæmia and intensity of the specific infection.

¹ Arch. f. Kinderh., vol. xxv. p. 321.

² Ztschr. f. klin. Med., 1902, vol. liv., Nos. 2 to 4.

³ Gaz. hebdomadaire de médecine, Mar. 18, 1900.

In the leucocytosis of syphilis some investigators have described a relative increase of the lymphocytes, others a slight relative increase of the eosinophiles.

Oppenheim and Löwenbach¹ have carried on a series of investigations in reference to the influence of mercury upon the various constituents of the blood in constitutional syphilis. They found that an anæmia with diminution of hæmoglobin and of iron was always present, but that the amount of hæmoglobin and the number of red blood-corpuscles corresponded to the iron contents of the blood, while the serum contained no iron, nor could any harm to the blood be demonstrated by the use of mercury in whatever form administered.

Seven years ago Justus described a blood-test for syphilis, which has since then been associated with his name. He described a marked diminution of hæmoglobin (ten to twenty per cent.) within twenty-four hours after mercurial inunctions or injections in all untreated cases of secondary, tertiary, and congenital syphilis, basing his claims upon the positive results obtained in three hundred syphilitic cases and the negative results in a large number of control experiments. Although Cabot and Mertens obtained similar results, the great majority of other investigators have arrived at absolutely opposite conclusions. Thus Brown and Dale, Jones, Christian, Foerster, Tucker, and Huger, all agree from their studies that the test is of no value, as the reaction is often absent in specific cases and is sometimes present in non-specific cases.

(8) *Pneumonia*. — In pneumonia a leucocytosis is regularly present, averaging, as a rule, between 15,000 and 30,000, while in the severe cases it may reach a much higher figure. Loehr reports a case in which 115,000 leucocytes per cubic millimetre were present. If the infection is very intense and the vital reaction practically nil, a leucopenia may be present, and the patient in this case almost always dies. With the crisis or shortly afterwards, the leucocytes fall to normal. Williamson² has studied the leucocytes in experimental pneumococcus infections in rabbits. He found, as a rule, a leucocytosis varying between wide limits. He did not observe an agonal leucocytosis, but was able to show that a diminished

¹ Deutsches Arch. f. klin. Med., 1901, vol. lxxi. p. 425.

² Ziegler's Beitr. z. path. Anat. u. z. allg. Path., vol. xxix., No. 1.

number of leucocytes was a distinctly unfavorable sign in pneumococcus infections.

The grade of leucocytosis tells us something of the severity of the infection, while if the leucocytes do not descend to normal with the drop in temperature, we know that the condition is a pseudo-crisis and not a true crisis.

(9) *Influenza*.—The normal condition of the blood in influenza, as regards the leucocytes, is a normal or sub-normal number of these cells. Blum,¹ from a study of the blood in this disease, finds that a leucopenia is especially likely to occur in the gastro-intestinal type of the disease, and believes that it is due to the involvement of the lymphoid tissues of the intestines and peritoneum. The absence of leucocytosis in influenza and the distinct diminution in the abdominal type of the disease are of value in differentiating influenza from lobar pneumonia, the catarrhal type of influenza from pertussis in the catarrhal stage, and the gastro-intestinal type of influenza from typhoid fever or from appendicitis.

(10) *Malta Fever*.—In Malta fever, as in most fevers of long course, there is an anæmia after the patient has been ill for a certain length of time, while Bruce, Müsser, and Sailer have found the leucocytes normal in most cases.

(11) *Gonorrhœa*.—A moderate leucocytosis is met with, as a rule, in gonorrhœa, although, in most of the cases, the increase is very slight and confines itself largely to the polymorphonuclear neutrophiles. Pozzoli² has made a careful study of the leucocytes in the blood and in the inflammatory exudate in gonorrhœa. He found that in anterior urethritis the gonococci were concealed in the crypts of Morgagni and in Littre's glands, and in posterior urethritis the gonococci were in the prostate in both acute and chronic cases. Eosinophiles were found in the secretion of the urethra and in the annexed glands. In chronic cases, the most eosinophiles were found in the prostatic secretion, a smaller number in the anterior and posterior urethra, and to the least extent in Littre's glands. The blood in the acute cases showed an inflammatory leucocytosis with a significant increase of the eosinophiles.

(12) *Plague, Cholera, and Yellow Fever*.—In bubonic plague,

¹ Wien. klin. Wchnschr., Apr. 13, 1899.

² Arch. f. Dermat. u. Syph., vol. xxiv., Nos. 1 and 2.

as a rule, the leucocytes are greatly increased. In yellow fever the leucocytes vary, although usually increased slightly, while degenerative changes are present in the red blood-corpuscles; while in Asiatic cholera the red blood count and leucocyte count are higher than normal, due in a large part, probably, to the profuse diarrhœa and the consequent concentration of the blood. In this last disease the blood may show a marked diminution of alkalinity or even a condition of acidity.

(13) *Chicken-Pox and Smallpox*.—The condition of the leucocytes varies in varicella, being sometimes normal, sometimes slightly diminished, sometimes slightly increased. In one case Nobécourt and Merklen report twelve and one-half per cent. of myelocytes, while if the vesicles become pustular, a neutrophilic leucocytosis of moderate degree has been described. In smallpox a leucocytosis has been described by many observers, as Hayem, Pick, Brouardel, and others, this leucocytosis reaching 35,000 per cubic millimetre in some cases, and being especially intense at the moment of vesiculation. Courmont and Montagard have shown that there is a marked increase of mononuclear forms in this disease, while Weil has found fifty-eight to sixty per cent. of mononuclears in the blood in this condition in seventeen cases of varioloid, while he also describes forms not normally met with, as plasma-cells, myelocytes, eosinophilic myelocytes, and basophiles. In hemorrhagic smallpox myelocytes have been met with in from ten to twenty-four per cent. Weil¹ believes that the contest between leucocytes and parasites takes place in the pustules, while in the severe hemorrhagic types of the disease the body seems to lose its defensive properties, as there is neither leucocytosis nor increase of fibrin in the blood.

Billings² has found a leucocytosis in vaccinated children beginning on the fourth day, reaching its maximum on the sixth to the eighth day, and falling to normal in from two to four days.

Sobotka also describes a leucocytosis after vaccination, beginning on the third or fourth day, lasting from three to five days; he also describes a second leucocytosis appearing from the tenth to the twelfth day, which Billings does not mention.

(14) *Epidemic Cerebrospinal Meningitis*.—Türk has described a leucocytosis of the polymorphonuclear neutrophilic type in this

¹ Gaz. d. hôp., 1901, No. 67.

² Med. News, Sept. 3, 1898.

disease, with a diminution of the lymphocytes, while the number of large mononuclears and transitional forms was unchanged.

(15) *Acute Articular Rheumatism*.—Acute articular rheumatism is usually accompanied by anæmia if the process has existed for any length of time, while a moderate leucocytosis of the ordinary type, averaging about 15,000, has been described during the febrile period by Stiénon, Achard, and Loeper; this leucocytosis may be much greater if complications are present. Achard and Loeper have described a similar kind of leucocytosis in gonorrhœal rheumatism.

(16) *Tonsillitis*.—Tonsillitis is practically always associated with a leucocytosis, according to Gilbert and Lion, the number of leucocytes varying between 9000 and 15,000. Jacob describes a slight eosinophilia at the close of the attack.

(17) *Other Infectious Diseases*.—In erysipelas the leucocytes are generally increased, for while Chantemesse and Rey found only from 7000 to 8000 in the mild cases, Hayem found 12,000 to 20,000 in the severe cases. The former observers found that the leucocytes, as a rule, rose and fell with the temperature. In cases terminating fatally, the leucocytes may be either greatly increased or sub-normal.

In mumps most observers describe a moderate leucocytosis with increase of neutrophiles and eosinophiles, and Sacquépée a leucocytosis of 8000 to 13,000 with a marked increase of the mononuclears of small and moderate size. If orchitis is present as a complication, the leucocytes are increased to a greater extent, this increase being largely confined to the neutrophiles.

In leprosy, Sabrazés and Mathis¹ found a constant eosinophilia in two cases of the nodular form of this disease, but in two cases of the anæsthetic form of the disease they found no changes whatsoever.

Achard and Loeper found five to eleven per cent. of the eosinophiles in leprosy, although Marie and Guilain found no increase of these cells in a severe chronic case.

In hydrophobia Courmont² has observed a marked leucocytosis in men and in animals, and in the animals experimented on, which

¹ Gaz. hebd. de méd., 1901, No. 14, and 1902, No. 2.

² Congress for Internat. Medicine, 1901, Centralbl. f. innere Med., 1901, p. 465.

included dogs, rabbits, and guinea-pigs. This leucocytosis he found to be present in the very beginning of the disease, and to continue until death; the increase is confined to the polymorphonuclear neutrophils, which frequently reach from ninety-five per cent. to ninety-eight per cent. Courmont regards this as a valuable diagnostic sign, especially in determining whether or not suspected animals are rabid.

(b) THE BLOOD IN DISEASES DUE TO ANIMAL PARASITES.

(1) *Malaria*.—Of course, the most important reason for blood examination in malaria is the examination of the blood, preferably a freshly prepared specimen, for the malarial parasite. As is well known, the three types of malaria, tertian, quartan, and æstivo-autumnal, are due to three distinct varieties of plasmodium. In the tertian form full growth is reached in about forty-eight hours, the corpuscle in which the plasmodium is found is larger and paler than the other corpuscles, the pigment-granules are fine, actively motile, and the number of segments into which the full-grown organism breaks up is usually between fifteen and thirty; in the case of quartan fever, the parasite takes about seventy-two hours to reach its full development, the parasite-containing corpuscle is smaller and deeper-colored than other corpuscles, the pigment granules are coarser and less actively motile, and the segments number from six to twelve; in æstivo-autumnal fever, only the very early forms, hyaline, ring-shaped, etc., are met with in the circulating blood at the beginning of the disease, as the further stages of development take place in the internal organs, especially in the spleen. According to different observers, either twenty-four or forty-eight hours are required for full development of the parasite. The corpuscle in which the parasite is found is smaller and of darker color than the normal corpuscle, and the number of segments usually vary between twelve and thirty; in the æstivo-autumnal infection, after a certain length of time, crescentic and ovoid forms make their way into the circulating blood and make the diagnosis easy. The blood always shows a diminution of red blood-corpuscles and hæmoglobin, the degree depending upon the duration and severity of the malarial attack, while almost all observers have described a diminution of leucocytes in this disease, although some have met with

a leucocytosis in a few cases where the anæmia was of an exceptionally high grade; Rogers, however, believes that the diminution of leucocytes stands in direct relation to the degree of the anæmia.

Vincent describes a leucocytosis lasting for a short period of time at the beginning of the paroxysm, while practically all observers agree that the typical blood picture is a marked diminution of leucocytes associated with a relative increase of the mononuclear forms, especially the large mononuclears according to most, but of the small mononuclears, according to Bastianelli and Vincent.

The diagnosis of malaria should never be made from the clinical history, but from a blood examination and the finding of the parasites; for in a number of conditions, such as syphilis, gonorrhœa, typhoid fever, pyosalpinx, and other suppurative processes, we may have a clinical picture almost identical with that of malaria, regularly intermittent temperature, chills, etc.; the presence of a leucocytosis in all these conditions, except typhoid fever, should be of great aid in making the differential diagnosis.

(2) *Filariasis*.—The diagnosis of filariasis is made by the discovery of the embryo of the *filaria sanguinis hominis* in the blood, these embryos ordinarily being found in the blood during the night. Calvert¹ has made most careful blood counts in four cases of filariasis, and finds regularly a moderate leucocytosis with an increase of the eosinophiles, these cells reaching twenty-two per cent. in one case. As the disease progresses, Calvert finds that the leucocytosis and eosinophilia gradually decrease to normal; he believes that the increase of eosinophiles is due to the local positive chemotaxis exerted by the embryos in the tissues upon the eosinophiles. Gulland and Coles have met with this same condition, while the former found that both leucocytosis and the proportion of eosinophiles increased during the night, when the embryos appeared in the circulating blood.

(3) *Diseases due to Intestinal Parasites*.—(A) *Trichinosis*.—In trichinosis T. R. Brown² describes a marked leucocytosis, reaching over 30,000 in some of these cases, with a marked increase of

¹ Johns Hopkins Hosp. Bull., Jan. and June, 1902.

² Ibid., Apr., 1897; J. Exper. Med., 1898, No. 3; Med. News, Jan. 7, 1899.

the eosinophiles, these cells reaching sixty-eight per cent. in one case, and calls attention to the diagnostic value of this blood picture, especially in differentiating this disease from other diseases associated with gastro-intestinal or muscular symptoms; in no other condition is such a high percentage of eosinophiles associated with such a marked increase of the leucocytes met with. Atkinson, Blumer and Newman, Stump, Kerr, Gwyn, Gordinier, Cabot, and others, have all diagnosed cases of trichinosis by this means, and it therefore seems highly probable that this blood phenomenon, marked leucocytosis with an enormous absolute and relative increase of the eosinophiles, is to be regarded as a definite diagnostic sign in this disease.

(B) *Anchylostomiasis*.—A very characteristic blood picture is presented in anchylostomiasis or uncinariosis, the disease due to the presence of anchylostomum (uncinaria) duodenale. The disease is characterized by a progressive anæmia often reaching a very high grade, the reds sometimes falling below one million per cubic millimetre, the anæmia being usually of the chlorotic type, while poikilocytes and nucleated red cells are also found. According to Ashford, megaloblasts are met with in one-fourth of the cases. According to Giles, Fernside, and others, the anæmia is due partly to loss of blood, partly to the damage done to the mucous membrane, and probably also partly due to some poison secreted by the worm. Giles¹ concludes that the color-index is always exceedingly low in this disease, being always below .4, while in uncomplicated malaria it is always above .5.

As to the condition of the leucocytes in this disease, practically all observers have met with a distinct increase of the eosinophiles in the blood associated with a marked increase of the eosinophiles in the intestinal excretions, here associated with the presence of the Charcot-Leyden crystals. Most observers found little or no absolute increase of the leucocytes, but Yates,² in a case studied by him, found 2,500,000 red blood-corpuscles and 24,000 white corpuscles, of which twenty-five per cent. were eosinophiles, while eight days later the reds had diminished to 800,000 and the whites were 29,600, of which but three per cent. were eosinophiles.

Zappert describes a case with seventeen per cent. of eosinophiles,

¹ Brit. M. J., Sept. 1, 1900.

² Johns Hopkins Bull., Dec., 1901.

while Leichtenstern met with seventy-two per cent. in one of his cases. As the number of cases met with in the United States will probably constantly increase, due to our close relationship with the Philippines and the West Indies, blood examination should always be made in cases of progressive anæmia associated with eosinophilia and intestinal symptoms.

(C) *Other Intestinal Parasites.*—In the case of a number of other parasites, an increase of the eosinophile-cells in the blood has been met with. In the case of oxyuris, Bucklers has had a case with sixteen per cent. of these cells, in the case of ascaris, nineteen per cent., and in the case of tænia, 8.75 per cent., while Leichtenstern has met with thirty-four per cent. in tænia, and Limasset with twenty-six per cent. According to Shaumann, eosinophilia is present in but a few cases of tænia bothriocephalus. A moderate eosinophilia has been described by many observers in the case of hydatid disease; Achard and Clerc found forty per cent. in one case, although it is usually of moderate degree, between four and ten per cent. Bezançon and Weil have inoculated the fluid from an hydatid cyst into guinea-pigs without obtaining any eosinophilia.

In a case of bilharzia hæmatobia, Coles found twenty per cent. of eosinophiles and forty-six per cent. of neutrophiles.

In the case of bothriocephalus, severe anæmia is frequently met with which is sometimes of the pernicious anæmia type and associated sometimes with an increase of the eosinophiles. Sawjalow¹ reports a case in which the red blood-corpuscles were 2,150,000 per cubic millimetre, and the hæmoglobin thirty-four per cent., and in which, after six weeks' treatment with potassium salts and sweating, a cure was obtained. The chemical examination of the blood showed an increase of the water contents, a diminution of the dried residue, and a markedly higher fat content than in normal blood. From the coincident increase of fats and extractives Sawjalow advances the hypothesis that pernicious anæmia may be a fatty degeneration of the red cells.

(c) THE BLOOD IN INFLAMMATORY PROCESSES, ABSCESSSES, APPENDICITIS, ETC.

Conditions coming under the especial Care of the Surgeon.—A leucocytosis is the condition regularly met with in all cases of inflam-

¹ St. Petersburg. med. Wehnschr., 1901, No. 27.

mation, suppuration, general or localized, appendicitis, peritonitis, cholecystitis, etc., except in the very mild cases, or in the most severe type of cases where the vital reaction is nil and the prognosis is almost invariably fatal. The type of the inflammatory leucocytosis is, of course, the polymorphonuclear neutrophilic. In localized suppurations, in septicæmia, and in pyæmia, leucocytosis is the rule; in the former case, it generally varies between 15,000 and 20,000, although it may reach much higher figures, as 50,000 and above, while practically the same figures are to be met with in septicæmia and in pyæmia.

We have seen several cases of generalized streptococcus or staphylococcus infections in which the leucocytes were markedly diminished, and death occurred in each of these cases, thus emphasizing the fact that leucopenia is an extremely grave prognostic sign in septicæmia.

Regarding the value of leucocyte counting in surgery, we feel that the question is no longer *sub judice*, for if we keep in mind the exact nature of leucocytosis and how it represents the conflict of two factors, virulence or infection and resistance of patient, we can derive a vast amount of knowledge from its study in surgical cases.

Da Costa,¹ from a study of one hundred and eighteen cases of appendicitis, found that in the simple catarrhal form of the disease the leucocytes remained within normal limits, while in the form associated with abscess formation, gangrene, or peritonitis, the leucocytes, as a rule, were markedly increased. A count of 25,000 or more per cubic millimetre points to the purulent form of the disease. According to Da Costa, the most important prognostic sign is that an absence of leucocytosis in obviously septic cases is very unfavorable.

Wasserman,² from a careful study of a large number of cases of appendicitis in which the leucocytes were carefully counted, concludes that the presence or absence of leucocytosis is the most reliable clinical sign we have in this disease, and may be the only method of determining whether or not operation is indicated.

Bloodgood,³ in a most carefully prepared paper upon blood

¹ Am. J. Med. Sc., Nov., 1901.

² München. med. Wehnschr., 1902, Nos. 17 and 18.

³ Maryland M. J., Sept., 1901.

examination as an aid to surgical diagnosis, concludes that in appendicitis the rising leucocytosis is an indication for operation, and in the majority of cases, if the leucocytes reach 18,000 before forty-eight hours, it is an indication of an advanced pathological lesion, as excessive exudate with a diffuse appendicitis, gangrene, appendix distended with pus, abscess, or beginning peritonitis. After the fourth day, a high leucocyte count was found to be associated usually with localized abscess or peritonitis, although, in abscess, the leucocyte count may be low in some cases. He found, in the majority of instances, that beginning peritonitis was associated with a rise in the leucocytes, which, however, rapidly decreased in number as the patient became more septic.

Bloodgood insists that in appendicitis leucocyte counts should be made every four to five hours during the first two days, and a rapid rise in leucocytes, especially above 18,000, should be a sufficient indication for operation.

Dunham¹ concludes that the leucocyte count in surgery is of the greatest value in diagnosing acute inflammatory conditions, and insists upon the importance of making two or more counts at intervals, this being especially useful in the early stages of appendicitis.

As influences which may cause leucocytosis in patients undergoing surgical treatment, he mentions a hearty meal, first years of life, hemorrhage, ether anaesthesia, the absorption of iodoform, carcinoma, and sarcoma.

Some surgeons, however, have come to different conclusions and pay but little attention to leucocyte counting. Thus, Hubbard,² from a leucocyte count in one hundred and eighty-nine non-inflammatory cases, in which the leucocytes averaged 8811 per cubic millimetre, seventy-one cases of inflammation not so great as to form pus in which the average was 12,645, and 299 cases of pus-formation with an average of 17,696, and in all three of which classes counts were found between 5600 and 25,800, concludes that as "most of the counts fall within these limits, they are of no practical value to the surgeon when brought face to face with an individual case, and from our present knowledge of the causes and variations of leucocytosis we cannot yet make any deductions which are simple enough to be of any great use to the surgeon at the bedside."

¹ Ann. Surg., June, 1900.

² Boston M. and S. J., 1900, vol. cxlii. p. 409.

We cannot agree with the conclusions of Hubbard, Deaver, and others, who ascribe but little practical value to leucocyte counting in surgery, and we feel that some, at least, have missed the vital point when they reach their conclusions. It is not so much the establishment of fixed rules between the grade of leucocytosis and the special pathological condition that is of great importance, but it is the result obtained by frequently counting the leucocytes in the individual cases to show whether the condition is lessening or progressing. Thus in appendicitis we feel that the leucocyte count is of great aid in determining whether we are dealing with an inflammation of the appendix or with a case of hysteria, floating kidney with twisted ureter, gall-stones, renal calculus, or the gastro-intestinal type of influenza. The grade of the leucocytosis will tell us much regarding the severity of the infection, while systematic counts at frequent intervals will show whether the condition is becoming better or worse, while in the case of children with appendicitis it is of especial value, due to the frequency in which the symptoms are masked in this condition.

Prognostically, in inflammatory and suppurative conditions, a diminution of leucocytes in a case with grave clinical symptoms may be regarded invariably as a bad sign, while after an operation the leucocyte count may be of great help in determining whether the condition presented is due to beginning peritoneal infection or to an autointoxication from the gastro-intestinal tract, in the latter of which conditions the leucocytes are but little, if any, increased. In intestinal obstruction, as a rule, the entrance of gangrene and localized peritonitis is shown by an increase of the leucocytes, while in differentiating abscess of the liver from malaria, duodenal catarrh, or impacted gall-stones; pyosalpinx from other pelvic conditions, or from malaria, or typhoid fever; or in diagnosing osteomyelitis from rheumatism, or neuralgia; tuberculous arthritis from other forms of arthritis; or to determine the presence or absence of secondarily invading micro-organisms in tuberculous processes, the leucocyte count is of great value. The presence of a marked leucocytosis associated with a diminution of red blood-corpuses, in the case of hemorrhage, is of great help in determining the cause of shock. Thus we feel that although it is impossible to formulate distinct and definite rules between surgical conditions, on the one hand, and leucocyte counts on the other, never-

theless much really practical aid may be derived from systematic blood counting in surgery.

(d) THE BLOOD IN CARDIOVASCULAR DISEASES AND NEPHRITIS.

Askanazy¹ furnishes an article of great value on the water contents of the blood and blood-serum in diseases of the circulatory organs, nephritis, the anæmias, and fever. On a careful consideration of the subject, Askanazy found that women have a lower specific gravity of the blood and less dried substance than men. On the other hand, he found, contrary to the view usually held, that women's serum was poorer in water and had a higher specific gravity than men's. In heart disease, if compensation was present, the blood as a whole and the serum were normal; if there was lack of compensation, the blood-serum showed a lower specific gravity, depending on the grade of the hydrops. The blood sometimes showed thinning, sometimes thickening, the latter condition being more marked where cyanosis was present in a high degree. The absolute water contents of the whole blood was independent of the grade of œdema.

In nephritis without œdema, normal blood was met with, with only occasionally a slightly increased water contents of the serum; if hydrops was present, the blood as a whole and the serum showed a not markedly increased water contents.

Both primary and secondary anæmias showed considerable increase of the water contents of the blood. In chlorosis the serum was usually normal, while other anæmias showed an increase of the water contents of the serum. In the case of the anæmias, the thinning of the blood, as a whole, is to be explained by a decrease of the red blood-corpuscles.

Stengel² discusses very exhaustively the blood in diseases of the cardiovascular system, and furnishes an extensive series of observations.

In acute endocarditis a rapid fall of the red blood-cells occurs, often to forty per cent. or fifty per cent., with an increase of the leucocytes, especially the polymorphonuclear neutrophiles, which may reach as much as 98.5 per cent. of all the forms. Often bac-

¹ Deutsches Arch. f. klin. Med., 1897, vol. lix., Nos. 3 and 4.

² Proc. Path. Soc. Phila., 1899.

teria are to be found in the blood, including the gonococci in a few cases.

In atheroma and chronic valvular disease fatty particles and detritus sometimes occur. In chronic valvular disease slight anæmia is sometimes present; in other cases, even with lack of compensation, an increase of 500,000 reds per cubic millimetre. In chronic cardiac disease, with continued slight inadequacy of the circulation, we often get polycythæmia, while in congenital heart disease, the red blood-cells may reach 8,000,000 per cubic millimetre. Grawitz explains this by loss of liquid from the blood, due to continually low blood-pressure and vascular dilatation. Stengel believes it to be due to a disturbance in the distribution of the corpuscles. In cyanosis an increased viscosity of the corpuscles is noted.

De Castelle¹ comes to the following conclusions regarding the influence of vascular changes and blood-pressure on leucocytosis:

(a) Strong stimulation of sensory nerves produces a leucopenia (diminution of leucocytes); (b) this result is dependent upon the reflex contraction of the abdominal vessels and the retention of leucocytes therein; (c) direct stimulation of the vasoconstrictor nerves of a certain territory causes leucopenia in the blood issuing therefrom; (d) the administration of vasoconstricting substances has the same effect, only in a higher degree; (e) the effects of shock (which causes leucopenia) depend upon similar vasoconstrictor phenomena; (f) the dilatation of vessels after section of the controlling nerves (splanchnics) has no influence upon the number of leucocytes; (g) reduction of blood-pressure produces only a transitory leucopenia; (h) in the experiments described the leucocyte count reaches its minimum in from three to four minutes, the original number being reached in from ten to sixteen minutes; (i) the marked leucocytosis in the splenic vein is due to the expression of leucocytes from that organ; (j) after the injection of substances producing leucopenia the leucocytes collect in the capillaries of the lung.

As a rule, in diseases of the kidney, with the exception of uræmia and hemorrhage, leucocytosis is not present, though in practically all conditions of nephritis secondary anæmia is met with.

¹ Wien. klin. Wehnschr., Apr. 13, 1899.

Lyon thinks that as carcinoma or sarcoma is usually associated with leucocytosis, and as malignant disease of the kidney is much more common than that of the spleen, the leucocyte count may be of value in differentiating malignant renal neoplasm from a non-malignant hypertrophied spleen, in which latter condition no leucocytic increase is met with, as a rule.

(e) THE BLOOD IN DISEASES OF NUTRITION AND METABOLISM.

(1) *Diabetes Mellitus*.—The most interesting work in connection with the blood in diabetes mellitus has been that done in connection with the Bremer and Williamson reactions, the latter of which is by far the more reliable. This reaction as described by Williamson¹ is as follows:

Forty cubic millimetres of water are placed in a small, narrow test-tube; to this are added twenty cubic millimetres of blood, one cubic millimetre of a one to six thousand aqueous solution of methylene blue, and forty cubic millimetres of liquor potassæ. The test-tube is placed in boiling water for four minutes, at the end of which time, if the blood is diabetic, the blue color will have disappeared and a dirty-green color will have taken its place. Williamson obtained the reaction in forty-three cases of diabetes tested, and thinks it is due to an increase of glucose in the blood. The reaction is of especial value in coma where urine cannot be obtained, and it can also be applied for a considerable time after death. Adler² concludes from a study of one hundred and thirty cases that the Williamson reaction is absolutely to be relied upon, but that the Bremer reaction is far less reliable; Adler believes that the reaction is due to a diminished alkalinity of the blood. Glycogen-granules are to be found in the plasma and in the leucocytes, while lipæmia is also sometimes present.

(2) *Gout*.—Garrod and others have found an increase of urates in the blood in gout, and the thread reaction, made by dropping a thread into the serum of the suspected cases, is used to show whether there is a uratic increase or not. Neusser thought that his perinuclear basophilic granules were characteristic of this disease and

¹ Phila. M. J., Jan. 14, 1889.

² Ztschr. f. Heilk., 1900, vol. xxi., No. 11.

were due to a deposition of uric acid in the leucocytes, but the work of Fletcher and Simon and others has shown that these granules are in no way characteristic of gout. Watson¹ has found myelocytes during the attack and in the intervals between the attack, especially in the former condition.

(3) *Purpura*.—In purpura, if associated with severe hemorrhages, a secondary anæmia and leucocytosis may be present, while in purpuric conditions the coagulation-time of the blood is frequently markedly increased, which is of extreme importance to the surgeon in deciding for or against the advisability of operation. Le Noble² discusses the subject of the clinical, prognostic, and pathogenic knowledge to be derived from a study of the serum in the case of the purpuras; this work he has carried on in association with Hayem for several years.

From this consideration he comes to the following conclusions: In Werlhof's disease the separation of the blood-cells from the serum is completely wanting, the hæmatoblasts show marked changes both in size and structure, and nucleated red corpuscles are constantly present; a slight leucocytosis and moderate anæmia are also present.

The same conditions are present in the severe chronic cases of purpura hemorrhagica.

If now the blood-cells and serum separate themselves partly, the hæmatoblasts are less markedly changed and nucleated red corpuscles less frequent, we are dealing with less severe forms of purpura, until finally we reach the forms in which the blood is normal (the rheumatic, infectious, scorbutic, and cachectic forms).

(f) THE BLOOD IN CANCER AND DISEASES IN THE GASTRO- INTESTINAL TRACT.

In the majority of cases of malignant diseases, wherever situated, a leucocytosis is present, which may appear early and constitute the very first sign of the disease, while it is often more marked in certain varieties of sarcoma than in the epitheliomas. Ordinarily the leucocytosis varies between 10,000 and 15,000, but is

¹ Brit. M. J., Jan. 6, 1900.

² Arch. prov. de méd., 1900, No. 9.

often much greater; thus Hayem found 52,700 leucocytes in a multiple osteosarcoma and 71,000 in a case of primary carcinoma of the thyroid. Strauss and Rohnstein found leucocytosis in sixty per cent. of the cases examined by them, while Hayem and Lion found 16,000 to be the average in cases of carcinoma of the stomach. The absence of the digestive leucocytosis in cases of carcinoma of the stomach is of great aid in diagnosis, although the same condition is met with in other non-malignant conditions, while Chadbourne has shown that digestive leucocytosis may be present sometimes in cases of gastric carcinoma. Strauss and Rohnstein, Bezançon and Labbé, found the neutrophiles increased, although in a few cases the eosinophiles showed the same result. According to Tuffier and Milian, the first leucocytosis in cancer is a mononucleosis, and the secondary polynucleosis is due to secondary infection with pyogenic micro-organisms. As a rule, in malignant leucocytosis there is no increase in the fibrin. Krokiewicz,¹ from a consideration of seventeen cases of gastric carcinoma, concludes that, though the blood is markedly changed in this disease, yet neither the changes in the red blood-corpuscles, the frequent absence of the digestion leucocytosis, nor the diminution of alkalinity, are pathognomonic of gastric carcinoma.

The author believes that a high count of the red blood-cells and a relatively high percentage of hæmoglobin, which remains stable during the course of the disease, speak for a secondary new formation of the growth on the peritoneum.

Osler and McCrae,² from a consideration of one hundred and fifty cases of gastric carcinoma, come to the following conclusions:

(1) In a doubtful case, a blood-count below 1,000,000 red blood-cells per cubic millimetre is strongly in favor of pernicious anæmia; (2) while nucleated red blood-corpuscles occur in all very severe anæmias, megaloblasts rarely, if ever, occur in cancer of the stomach; (3) neither an increase in the leucocytes nor special variations in the various forms appear to be of any moment in diagnosis of gastric carcinoma; and (4) the presence or absence of a digestion leucocytosis is too uncertain to be of much assistance in diagnosis.

¹ Arch. f. Verdauungskrankh., vol. vi. p. 25, and Wien. klin. Wchnschr., vol. xii., No. 37.

² New York M. J., May 19, 1900.

We have already called attention to the fact that, as a rule, the sugar in the blood is increased in carcinoma, but not in sarcoma.

(g) THE BLOOD IN MENTAL AND NERVOUS DISEASES.

Zappert found, as a rule, a moderate increase of the eosinophiles in the organic and functional nervous diseases, while Neusser describes their increase in a variety of nervous disorders without, however, giving the figures of his differential counts.

(1) *General Paralysis*.—Capps,¹ from a study of the blood in twenty cases of general paralysis, found that the hæmoglobin and red blood-corpuscles were always decreased, and that the specific gravity was always slightly diminished. There was usually a slight leucocytosis, which was absent in the early stages of the disease. The lymphocytes were diminished, the large mononuclears increased. In a few cases the eosinophiles were very numerous. At the time of a convulsion the red blood-cells and hæmoglobin were increased, while during an apoplectic attack of longer duration they were diminished. After convulsions and apoplectic attacks, a leucocytosis would make its appearance, the grade of which depended directly upon the severity and length of the attack; here the large mononuclears were increased.

Jenks² concludes from a study of the white blood-cells in nine cases of general paralysis that there is an increase of large mononuclears prior to epileptiform and congestive seizures, but not especially at other periods, and that the increase of these cells justifies a suspicion of the attack.

(2) *Chorea*.—Burr³ and Murphy⁴ have found that in the vast majority of cases of chlorosis the red blood-corpuscles and hæmoglobin become somewhat diminished, while T. R. Brown,⁵ from twelve observations on cases of chorea, found a moderate eosinophilia, usually between five and nine per cent., always present.

(3) *The Functional Neuroses*.—Lunenburg⁶ has studied the blood changes carefully in the functional neuroses, and finds that

¹ Am. J. M. Sc., vol. cxi. p. 650.

² Am. J. of Insan., Jan., 1900.

³ Univ. M. Mag., vol. ix. 3, p. 183.

⁴ Kansas City M. Rec., Dec., 1899.

⁵ Maryland M. J., July, 1902.

⁶ Centralbl. f. innere Med., 1899, No. 21.

(1) the blood, contrary to the usual view, shows no anæmic symptoms, that is, no hydræmia, but normal water contents; (2) the red blood-corpuscles are often distinctly increased, probably due to the ease with which vasomotor disturbances may be set up at the time of the examination; (3) the leucocytes are often subnormal; (4) marked precipitating changes often take place.

(4) *Beriberi*.—Mott and Halliburton¹ from the blood in a case of beriberi obtained a substance causing a distinct fall of blood-pressure, while in the acute stages of the disease eosinophilia has been described.

(5) *Diseases Associated with Convulsions*.—Burrows² has studied the blood in seven cases associated with convulsions, two of senile dementia, two of general paralysis, one of katotonia, one of puerperal eclampsia, one of terminal dementia, besides two cases without convulsions, one a general paralytic, the other a normal and athletic young man. These last two, the first in his frenzies, the second in his exercises, showed a leucocytosis, but of low degree, of short duration, and not of the inflammatory type, that is, the percentages of the different varieties of leucocytes were normal, while in the seven cases associated with convulsions, the leucocytosis was of the inflammatory type, that is, the polymorphonuclear neutrophiles were markedly increased, often reaching ninety-five per cent., while in some of these cases the leucocytosis reached 50,000. Thus there is a leucocytosis associated with convulsions, not only in general paralysis, but in other cases as well, and the grade of this leucocytosis bears a relation to the severity of the attack.

(6) *The Blood in other Mental and Nervous Diseases*.—In epilepsy, Krainsky³ found that the carbamic acid in the blood was considerably increased, and that this increase was in direct relation to the violence of the epileptiform attacks, and the same was true of the ammonia in the blood; but Herter⁴ found that in no case was there any evidence that in epilepsy the blood was more toxic than normal blood.

¹ Brit. M. J., July 29, 1899.

² Am. J. M. Sc., May, 1899.

³ Allg. Ztschr. f. Psychiat., vol. liv. 4, p. 612.

⁴ J. of Nerv. and Ment. Dis., Feb., 1899.

(h) THE BLOOD IN PREGNANCY, LABOR, AND THE PUERPERIUM.

We have already stated that regularly in the case of primiparæ and frequently in the case of multiparæ a leucocytosis is met with during the latter months of pregnancy, while in the puerperium the same condition is also frequently met with.

Carstanjen¹ has studied the blood in a large number of cases in the latter days of pregnancy and the early days of the puerperium, with the following results:

During the latter days of pregnancy the percentage of neutrophiles, lymphocytes, transitional forms, large mononuclears, leucocytes, and eosinophiles averaged 69.31, 18.62, 9.47, 0.23, and 2.37, respectively; one day after delivery, 78.9, 13.56, 5.63, 0.29, and 1.62, respectively, while one week after delivery they averaged 66.56, 20.52, 9.2, 0.52, and 3.2, respectively.

Hibbard and White² found a leucocytosis during labor and the puerperium in all the fifty-five cases studied by them. This leucocytosis reached its maximum at the time of labor and was most marked in young women, especially primiparæ, while Biegonne³ noted a general increase in the large mononuclears during the last days of pregnancy, while on the day after delivery there was an extraordinary increase of leucocytes, all forms being effected. Rieder gives 13,000 as the average number of leucocytes during pregnancy, but Ascoli and Esdra⁴ did not find this except in the last month of pregnancy.

(i) THE BLOOD IN DISEASES OF THE SKIN.

In diseases of the skin many observers have noted an eosinophilia in certain cases. Thus it has been described in pemphigus, eczema, scleroderma, psoriasis, pellagra, lupus, and urticaria, while a local eosinophilia is quite frequent in many cases where there is no general increase of these cells in the blood. Brown and Dale⁵ found twenty-nine per cent. of eosinophiles and a leucocytosis varying from 9000 to 14,000 in a case of dermatitis herpetiformis,

¹ *Jahrb. f. Kinderh.*, vol. lii., Nos. 2 to 4.

² *J. of Exper. Med.*, 1898, vol. iii., No. 6.

³ *Arch. russe de Path.*, etc., 1898, vol. vi. p. 70.

⁴ *Bull. d. Soc. Lancisiana*, 1898, vol. xviii. p. 2.

⁵ *J. Am. M. Ass.*, Feb. 17, 1900.

while Okamura¹ found a marked leucocytosis averaging over 40,000 in three cases of xeroderma pigmentosum, while in two of these a moderate eosinophilia was also present.

Colombini² and T. R. Brown³ describe a moderate leucocytosis with about ten per cent. of eosinophiles in epidermolysis bullosa hereditaria.

Eosinophilia seems to be especially common in skin-diseases associated with vesicles, as pemphigus, Hallopeau's dermatitis, Duhring's disease, and various forms of herpes, while Sabrazés and Mathis describe a leucocytosis with eosinophilia in herpes zoster.

Zollikofer⁴ found that after the use of various vesicants there was a slight increase of the leucocytes in the general circulation and in the blood-vessels of the part.

Achard and Clerc⁵ report an interesting case in which after the use of a few drops of picric acid applied externally a general scarlatiniform dermatitis broke out with vesicles on the face, hands, and feet, and this was associated with an eosinophilia of fifteen per cent.

The eosinophilia met with in the skin-diseases is of especial interest, because of the fact that Ehrlich believes that the substances produced by the disintegration of epithelial cells have a marked chemotactic effect upon the eosinophiles.

(j) THE BLOOD IN VARIOUS INTOXICATIONS.

(1) *Lead Poisoning*.—We have already called attention to the fact that one of the earliest characteristics of the blood in lead poisoning is the appearance of basophilic granules in the red blood-corpuscles. These granules have been regarded as evidences of cellular degeneration by Hamel, Kohn, Grawitz, Stengel, and others. In chronic lead poisoning, an anæmia is regularly present. Achard and Loeper describe a slight neutrophilic leucocytosis in three cases of lead colic.

(2) *Poisoning with Coal-Tar Preparations*.—A leucocytosis has been described in a large number of cases after the use of many

¹ Arch. f. Dermat. u. Syph., Jan., 1900.

² Monatsch. f. prakt. Dermat., May 15, 1900.

³ Maryland M. J., Apr., 1901.

⁴ Deutsches Arch. f. klin. Med., vol. lxxix., Nos. 3 and 4.

⁵ Gaz. hebd. de méd., 1900, No. 81.

of the coal-tar derivatives, often associated with a rapidly produced anæmia if the doses are large. Thus Ehrlich describes this condition after the derivatives of phenyl-hydrazin and phenacetin, while Achard and Loeper describe it after antipyrin, and T. R. Brown after acetanilid, in which last mentioned case 30,000 leucocytes with twelve per cent. of eosinophiles were met with in a case of acute poisoning.

(3) *Poisoning with other Substances.*—A leucocytosis has been described after the use of an enormous number of other poisons, medicines, etc.; thus, Chadbourne¹ reports twenty-one cases of leucocytosis after the administration of ether, although Benassi² describes a decrease of leucocytes after chloroform anæsthesia. After tuberculin a marked eosinophilia, usually appearing after the fever has diminished, has been described by Zappert, Ehrlich, and Lazarus, while Grawitz, three weeks after a series of injections of tuberculin, found that, of 45,000 leucocytes per cubic millimetre, the majority were eosinophiles. Leredde describes an eosinophilia after the use of camphor. Among the other substances which frequently produce a leucocytosis may be mentioned potassium chlorate, alcohol, mercury, snake-venom, and also antivenene, nucleinic acid, various of the bitter tonics, ethereal and essential oils, and a number of other substances, although, of course, the leucocytosis is by no means a constant accompaniment of these substances. An interesting piece of work in this connection is that of Bentvegna and Carin, who found in a series of animal experiments that after doses of arsenic, iodine, and bichloride of mercury which were not fatal, a marked leucocytosis was found, while in the fatal cases a diminution of leucocytes occurred, and, with this, a diminution in the bactericidal powers of the blood.

(k) THE BLOOD IN VARIOUS OTHER DISEASES AND PATHOLOGICAL CONDITIONS.

(1) *Diseases of the Internal Genital Organs in Women.*—Voswinckel³ has studied the blood carefully in diseases of the female internal genital organs.

¹ Phila. M. J., Feb. 18, 1899.

² Gazz. d. osp. e delle clin., 1901, No. 21.

³ Monatschr. f. Geburtsh. u. Gynaek., vol. vii., No. 4, p. 413.

One hundred and twenty-six cases in all were examined, with the following results:

The eosinophiles and myelocytes were normal in tubal disease, uterine myomata, and endometritis; in severe but not feverish or malignant diseases there was an eosinophilia constantly, and often also the presence of myelocytes. In two cases of severe ovarian disease with fever, and in two cases of uterine carcinoma, there was an eosinophilia, while in cystic conditions of the ovaries or diseases associated with pus-production, tending to a complete degeneration of the ovarian tissue, in ten of eighteen cases there was eosinophilia and an increase of myelocytes.

(2) *Bronchial Asthma*.—In bronchial asthma a marked increase of the eosinophiles in the blood, reaching sometimes to twenty or thirty per cent. or even more, associated with the appearance of large numbers of the eosinophiles and Charcot-Leyden crystals in the sputum, has been described by a number of observers, especially Gollasch, Fink, and Gabritschewsky. While this increase is most marked during the paroxysm and just afterwards, nevertheless in a number of cases a moderate eosinophilia has been met with for a long period after an attack. The eosinophilia in bronchial asthma may be of use in differentiating it from cardiac or renal asthma.

(3) *Thyroidectomy*.—After experimental thyroidectomy in dogs Levy¹ found, as a rule, a marked anæmia which usually appeared early. A leucocytosis was also found, the number varying between 32,000 and 44,000, while many small forms made their appearance on the day following the operation. Mezincescu found that the same condition occurred in human beings after thyroidectomy, the leucocytes in one case reaching 49,000, with eighty-four per cent. of neutrophiles.

(4) *Jaundice*.—In jaundice, as we have said before, the bile-pigment is present in the circulating blood, where it may be demonstrated by the usual reactions and the coagulation time may be much increased.

(5) *Addison's Disease*.—In Addison's disease there is usually a secondary anæmia if the condition has been present for any length of time, while as to the changes in the leucocytes, although they are generally described as normal, Cabot describes one case with 14,000 leucocytes and four and one-half per cent. of eosinophiles, T. R.

¹ Brit. M. J., Sept. 3, 1898; J. Path., 1898, vol. iii. p. 316.

Brown two cases, one with 6500 leucocytes and four and four-tenths per cent. of eosinophiles, the other with 12,000 leucocytes and eight per cent. of eosinophiles, in this latter case there being but twenty-nine per cent. of hæmoglobin.

(XII.) SERUM DIAGNOSIS, SERUM THERAPY, AND IMMUNITY.

(a) GENERAL CONSIDERATIONS.

No article upon the blood would be complete without a consideration of those most interesting medical questions, serum diagnosis, serum therapy, and immunity; and yet the amount of work done upon this subject has been so enormous that in this article we shall have to content ourselves with a brief consideration of the modern views held regarding this subject, followed by a rapid *résumé* of some of the recent articles on the serum diagnosis and the treatment by sera of the various special diseases.

Therefore, after the brief general consideration, we shall take up in turn typhoid fever, diphtheria, tuberculosis, tetanus, pneumonia and pneumococcus infections, cholera, plague and yellow fever, Malta fever, relapsing fever, leprosy, colon-bacillus infections, infections with the bacillus pyocyaneus, with the bacillus proteus and the vibrio septicus, anthrax, infections due to the streptococcus, pertussis, malaria, and snake-poisoning.

Gay¹ furnishes a very well-written and clear *résumé* of the modern theories regarding toxins and antitoxins as elaborated by Ehrlich, Bordet, Wassermann, and others.

Toxins are labile, poisonous substances, elaborated by plant and animal cells, of which we have no exact chemical knowledge, but whose characteristics are recognizable by their physiological effect on animals. Toxins differ from all known chemical poisons in these important particulars:

- (1) They are subject to cell assimilation, that is, they enter into direct chemical combination with protoplasm, whereas chemical substances either destroy the cell or are present in it in solution.
- (2) Toxins can call forth the production of antitoxins in the animal body, while chemical poisons cannot.
- (3) The action of toxins is delayed by a considerable incubation period.

Toxins which have undergone change by time or certain chemi-

¹ Am. J. M. Sc., May, 1902.

cal substances form substances known as toxoids, which possess an equal power of stimulating the formation of antitoxins, but have relatively little toxic effect.

Antitoxins are substances formed in the animal body by the inoculation of toxins or toxoids; they are recognized by their action in neutralizing the effect of toxins.

The variation in duration of active and of passive immunity, the presence of normal antitoxic bodies, and the increased production of antitoxin under the action of pilocarpine, are all observations which make it certain that antitoxins must be regarded as products of cell activity.

Virchow pointed out in his "Cellular Pathology" that chemical substances were picked out by certain organs. The almost absolute absorption of the toxins of tetanus and diphtheria by nerve-cells are further instances of the elective action. The obvious example of the variation of leucocytes in their reaction to stains is but a further step in cell differentiation. It is on this basis of finer cell structure, as suggested to us, that Ehrlich has conceived each cell as possessing, in addition to its nucleus, multitudes of atomic "lateral chains" or "receptors," each with its affinity for a certain assimilable substance. The normal function of the receptors lies principally in this choice of suitable foodstuffs, but toxins as assimilable substances are also taken up by receptors fitted for this purpose.

The toxin unit must now be considered as more than a simple body; it possesses, in fact, at least two parts, a "haptophore" group, which unites it to the cell receptor, and a "toxophore" complex, which is the poisonous element. Toxoids as altered toxins possess only the haptophore group, which accounts for their absence of toxic effect. The body-cell is more or less injured if the united unit be a toxin, owing to the toxophore group; but even if there be no toxophore present, as in a toxoid, the receptors united are thrown out of function. Loss of the cell constituents in either case leads to regeneration of the parts affected, and regeneration of tissues, as Weigert has shown, takes place always in excess. This excess of toxin elective "receptors" cumbers the cells, and they are eventually thrown off into the circulating blood, where they form the antitoxins. A toxin cannot harm a cell unless its haptophore group unites with a cell "receptor;" when "circulating

receptors" or antitoxins are present in the blood the haptophore groups become anchored and never reach the cell, which accounts for the neutralization of the action of toxin by antitoxin. Absence of "receptors" suitable to unite with toxins explains the natural immunity of certain animals to certain toxins. Predominance of suitable receptors accounts for the election of certain toxins by certain tissues. A multiplicity of toxic bodies such as may occur in a single bacterial toxin simply calls for a corresponding multiplicity of receptors.

The period of incubation is the time during which the "receptors" are regenerating in the cells and before they are thrown off into the circulation. That the origin of antitoxins is primarily in the fixed cells is further shown by the fact that organs acquire anti-toxic properties before the blood itself.

"Receptors" which unite with toxins are called "receptors of the first order," being relatively simple and having only one receptive process. Similarly toxins are "uniceptors," that is, they have only one uniting arm, the haptophore group. This conception is better understood on comparison with the structure of the hæmoly-sins and their "receptors."

Welch's magnificent Huxley lecture for 1902 should be read by every one interested in this subject.

The amount of work done to determine the relation of toxins to antitoxins, etc., has been enormous, and it would carry us far beyond the limits of our paper to attempt to mention more than a few of the articles of interest in this connection. Pawlowsky¹ discusses the fate of various micro-organisms in the bodies of susceptible and of immune animals, the bacteria being made to experimentally enter the body through the skin and subcutaneous tissues, that is, by the route in which infection is usually introduced into the body. The bacteria used by him in this experiment were staphylococcus aureus, bacillus pyocyaneus, typhoid and diphtherial bacilli, etc.; both susceptible and immune animals were experimented upon. From his experiments he concludes that the bacteria introduced under the skin soon reach the blood and the internal organs by way of the lymphatics, and many of them are eliminated in the urine and bile.

¹ Ztschr. f. Hyg. u. Infektionskrankh., vol. xxxiii. pp. 261-312.

In artificially immunized animals the bactericidal, agglutinating and antitoxic bodies are very unequally distributed in the different organs and tissues, and even in normal organisms there is a very unequal distribution of the antitoxins present. For the pyogenic micro-organisms the bone-marrow and the spleen are the organs which apparently are most important in their destruction, harmonizing with Ehrlich's and Wassermann's views of immunity in tetanus and typhoid.

Halban and Landsteiner¹ have contributed an article of interest in connection with the differences between the serum of the new-born and the serum of the mother. Already it has been shown that at the moment of birth the blood of the new-born child is poorer in fibrin and richer in formed elements than the maternal blood (Krüger), that the erythrocytes are less resistant than those of the mother (Scherenzius, Doléris, and Quinquaud), and that its cryoscopic point is lower than in the maternal serum (Veit).

Halban and Landsteiner's experiments have been conducted to determine the differences between foetal and maternal serum in regard to agglutinating, hæmolytic, and bactericidal properties, and their results are as follows:

(1) The serum of the mother and that of the child do not react in the same way. The hæmolytic, agglutinating, bactericidal, antifermentative, antitoxic, and precipitating properties are much more marked in the maternal serum than in that of the new-born child.

(2) These facts show that the active substances of the serum are present in the new-born, but that they are in smaller quantity and act less energetically than in the adult. These differences explain the diminished resistance which an infant has towards infections and intoxications.

The results of Castellani's experiments² upon agglutination in mixed infections, the typhoid bacillus, colon bacillus, and pseudo-dysenteric bacillus being employed, were as follows: In the case of a mixed experimental infection the serum of the animal agglutinates all the micro-organisms which have produced the infection. In the case of each of these micro-organisms the agglutination pre-

¹ München. med. Wehnschr., Mar. 25, 1902.

² Ztschr. f. Hyg. u. Infektionskrankh., May 2, 1902, vol. xl. p. 1.

sents the same characteristics as to its beginning, intensity, and duration, as in those cases where the infection has been produced by a single species. When in the course of a monomicrobial experimental infection another micro-organism is injected, the animal serum becomes agglutinating towards this micro-organism, and the agglutination possesses the same characteristics as in the case of a monomicrobial infection; sometimes the agglutination of the second micro-organism appears more slowly and less energetically if the injection is made during the course of the first infection.

Walger¹ discusses therapy with the blood-serum of convalescents in the treatment of acute infectious diseases. He reports a number of cases so treated, and calls especial attention to the great improvement in the general condition of the patient immediately after the serum injections, although the complications that may be present may take a considerable time to disappear. This method of treatment will be again referred to many times during the course of our consideration of serum therapy in special diseases and pathological conditions.

(b) TYPHOID FEVER.

In typhoid fever we may conveniently divide our considerations into two parts: (1) The diagnosis of typhoid fever by means of the Widal reaction, and by the bacteriological examination of the blood, and (2) the treatment of typhoid fever by serum therapy, vaccination, and the use of the organs and tissues from animals or from human beings suffering from typhoid infections.

The value of the Widal reaction is, of course, too firmly established to need any discussion, but the time at which the reaction first makes its appearance and the duration of the reaction are still worthy of a short consideration.

Van Houtum² found that of ninety-seven cases of typhoid at the hospital at Rotterdam twelve did not give the Widal reaction during the second and third weeks. Of these twelve, four died during this period, while the remaining eight all gave the reaction during the fourth week. In examining thirty cases that did not have typhoid fever, agglutination occurred in seven in a dilution of one

¹ *Centralbl. f. innere Med.*, 1898, Nos. 37 and 49, and 1902, No. 7.

² *Nederl. Tijdschr. v. Geneesk.*, 1898, vol. ii. p. 840.

to ten, in six in one to twenty, in three in one to thirty, while in stronger dilutions the agglutination was not observed either microscopically or in the hanging drop.

Tobiesen¹ found that in three hundred and fifty cases of typhoid fever a positive reaction in a dilution of one to fifty occurred in three hundred and twenty-nine cases, while in seventeen, although the reaction was positive at one to twenty-five, it was always negative at one to fifty. Only one case agglutinated in this series in a dilution of one to fifty one year after the attack.

Withington² and Shattuck³ report their experiences with the Widal reaction. The former in a series of two hundred and fifty-nine cases reported six per cent. of failures, the latter had three failures in sixty-two cases.

Zupnik,⁴ from his observations in two hundred and three cases of typhoid fever, believes that a positive reaction signifies undoubted typhoid, if a previous attack of the disease or the presence of jaundice can be ruled out. Of other cases examined, and these included cases of miliary tuberculosis and sepsis, only those cases associated with jaundice gave a positive reaction.

Gershel⁵ obtained a positive Widal reaction in eighty-one of eighty-four cases in children from eighteen months to fourteen years of age, while in one hundred and fifteen cases of other fevers the results were all negative.

The only objection to the Widal reaction has been the fact that it usually does not make its appearance until the ending of the first or beginning of the second week, while it often appears much later still. On this account various methods have been suggested by means of which an earlier diagnosis may sometimes be made. Of these, the examination of the blood for typhoid bacilli offers the greatest hope of success.

Cole⁶ used the following technic in making blood cultures in typhoid fever: A bichloride compress was kept on the elbow (internal aspect) for one hour, after which the arm was washed with

¹ *Ztschr. f. klin. Med.*, 1901, vol. xliii., No. 1.

² *Boston M. and S. J.*, 1901, No. 19.

³ *Ibidem.*

⁴ *Ztsch. f. Heilk.*, 1901, No. 11.

⁵ *Med. Rec.*, Nov. 26, 1901.

⁶ *Johns Hopkins Hosp. Bull.*, July, 1901.

sterile water, and, by means of a cannula, ten cubic centimetres of blood were removed from the vein. This was transplanted in bouillon, and after twenty-four hours from the bouillon on agar plates. By this means the diagnosis can be reached in thirty-six hours, but if a more rapid result is desired, six to eight hours after the agar culture has been made, a suspension of this culture in bouillon can be used with typhoid serum as a test for the micro-organism.

Typhoid bacilli were obtained in eleven out of fifteen cases tested by this method, generally during the second week, once on the sixth day. The cultures were positive in cases where no Widal reaction was obtained from the serum. In six of twelve cases tested typhoid bacilli were also found in the urine.

Hewlett¹ found the typhoid bacilli in the blood in twenty cases of typhoid fever of twenty-four studied. In three cases they were found before the Widal reaction was positive. Their earliest appearance was on the fourth and fifth days of the disease, while they disappeared from the blood when the temperature began to fall.

Busquet² has collected upwards of two hundred cases of typhoid fever, including forty-two of his own, in which the bacteriological examination of the blood was made, in about seventy per cent. of which the results were positive. The first observation of this kind was made by Frenkel and Simmonds in 1885.

In eight of Busquet's forty-two cases typhoid bacilli were demonstrated in the blood before the appearance of the Widal reaction. He regards it as a most valuable procedure, not only as an aid in making the diagnosis of typhoid fever, but in those cases which clinically present the symptoms of typhoid fever, but are due to other micro-organisms.

The specific treatment of typhoid fever has been carried on upon three distinct lines during the past three years: First by serotherapy, second by anti-typhoid inoculations, and third by the use of extracts of organs from animals experimentally inoculated with the typhoid bacillus.

Chantemesse³ gives a most careful review of his work in the treatment of typhoid by the use of serum prepared from horses

¹ Med. Rec., Nov. 30, 1901.

² Presse méd., June 21, 1902.

³ Ibid., Nov., 1901.

artificially inoculated, which work he has been carrying on since 1892 with Widal. By the use of this serum fifty-five cures were obtained in fifty-nine cases of typhoid fever so treated, while among the typhoid cases in the same hospital not so treated the mortality was between twenty and thirty per cent. Only patients who were seriously or gravely ill were given injections. All were cured who were treated before the tenth day; of the one hundred treated by him by this method six died, three of perforations, one of lumbosacral abscess, while in one case the injection was not given until the twenty-first day of the disease.

The chances of cure are good if the injection is given before the eighth day of the disease, while one may hope for a rapid deferrescence if it be put off until the ninth to the twelfth day. All such influences as alcoholism, syphilis, fever of long duration, or an intense and generalized infection tend to make the effect of the serum less marked, and in cases of this nature a larger dosage or a second injection is recommended. In a certain number of cases relapses occur from eight to twelve days after the injection, due to the fact that all the germs are not destroyed nor their toxins neutralized by the first injection. On the least appearance of a second rise of temperature an inhibiting dose of the serum should be given immediately. The influence of the treatment is very marked, notably diminishing the duration of the disease, increasing the blood-pressure, lowering the pulse-rate, while all the other symptoms are markedly improved. The blood is remarkably modified, as after the injection it shows the changes normally met with at the conclusion of an attack of the disease, that is, leucocytosis, the reappearance of the eosinophiles, and an increase in the transitional cells and polymorphonuclear leucocytes. The serum has been obtained from horses which had been vaccinated since 1896, for an attempt to prepare the serum more rapidly often kills the horse experimented upon.

Von Jaksch¹ used the blood-serum of patients convalescent from typhoid in the treatment of this disease, without noticing any favorable results, which agrees with the results obtained by Jez.²

¹ *Verhandl. d. XIII. Cong. f. innere Med., Wiesb., 1895, p. 539.*

² *Wien. med. Wehnschr., 1898, vol. xlviii. No. 19.*

Wagner,¹ however, thinks that in four cases so treated by himself distinctly favorable results were noted.

Various methods have been devised as a protection against typhoid fever, in other words to immunize exposed individuals, but among these none has been carried on so carefully, or on such a large scale, as the experiments of Wright and others in the British army.

Duckworth² describes the method of procedure in using the antityphoid vaccine, which was kindly furnished him by Wright. One cubic centimetre of the vaccine was given, and in two hours the temperature had risen two degrees, and the patient complained of slight headache; the temperature remained above normal for forty-eight hours. A second inoculation was given nine days later, followed by the same symptoms, but of a milder grade. A week after the second injection, the blood agglutinated the typhoid bacillus in a dilution of one to two hundred.

This method has been employed to a great extent in the British army, and while it will be impossible to consider all the results reported, some of the figures in this connection are extremely interesting.

Wright³ considers the result in the cases of the XVth Hussars, stationed in India, and of the English troops stationed in Cyprus. Of the former, three hundred and thirty-six were inoculated, of whom two were taken ill with typhoid, and one died, while of one hundred and sixty-seven comrades not inoculated, eleven contracted typhoid, and six died; of the latter, seven hundred and twenty soldiers were inoculated, of whom only one contracted typhoid, while of two thousand six hundred and sixty-nine comrades, who were not inoculated, sixty-eight contracted typhoid, of whom ten died.

Tooth⁴ gives his results in the Orange Free State. Of two hundred and thirty-two cases of typhoid, fifty-four had previously received preventive inoculations, and of these seven and four-tenths per cent. died, while of the one hundred and seventy-eight not so treated fourteen per cent. died.

¹ *Centralbl. f. innere Med.*, 1900, p. 941.

² *Lancet*, 1899, vol. ii. p. 1407.

³ *Ibid.*, Feb. 9 and May 4, 1901.

⁴ *Ibid.*, Mar. 16, 1901.

Cayley¹ gives the results of the inoculations at Kronstadt and Bloemfontein during a severe typhoid epidemic. Of the sixty-one persons constituting the first section of the hospital corps, fifty-seven were inoculated twice at an interval of ten days, two orderlies were inoculated once, while two nurses, who had previously suffered from typhoid, were not inoculated. This section had the care of typhoid cases in large number and of great severity, but none contracted the disease. Four months after the inoculation the Widal test was tried in the case of twenty-three of these persons; the reaction was prompt and characteristic in twenty-one of these in dilutions of from one to forty to one to five hundred, but the two orderlies, who were inoculated but once, gave only slight reactions. In the second and third sections of the hospital equally satisfactory results were obtained in those who had been inoculated twice, but one inoculation did not seem to be sufficient in all cases, as a few of the nurses and orderlies inoculated but once contracted the disease.

Wright² has furnished recently a most interesting article containing his views on the changes effected by antityphoid inoculations on the bactericidal power of the blood. The following practical conclusions are reached by him as to the mode of administration, the quantity to be given, and the effect of antityphoid inoculation:

1. The employment in primary inoculation of large doses of vaccine—meaning thereby doses sufficient to give rise to very severe constitutional symptoms—would appear to be always inadvisable, while it would probably be associated with danger in cases where inoculation is resorted to in the actual presence of a typhoid epidemic.

2. The employment of moderate doses of vaccine—meaning thereby doses sufficient to give rise to marked, but not severe, constitutional symptoms—would appear to be inadvisable when making primary inoculations in the actual presence of a typhoid epidemic. On the contrary, such doses would seem to be appropriately employed where an interval of several weeks is to elapse before exposure to infection, and where there are difficulties in the way of carrying out two successive inoculations.

3. The employment of small doses of vaccine—meaning thereby

¹ Brit. M. J., Jan. 12, 1901.

² Lancet, Sept. 14, 1901.

doses which produce only a slight constitutional disturbance—would appear to be the only appropriate form of inoculation in the actual presence of typhoid infection. It would seem to be also in all other cases the most appropriate form of inoculation. Such primary inoculation ought, however, in all cases to be followed up by second inoculations with an increased dose of vaccine.

The treatment of abdominal typhoid with extracts prepared from the organs of animals inoculated experimentally with the typhoid bacillus was originally used by Jez. His method of preparing this extract¹ is as follows:

Guinea-pigs were given intraperitoneal injections of typhoid cultures of steadily increasing virulence, until tolerance was established, when the animal was killed and an extract made of the various organs by cutting them into pieces of small size and rubbing them up with a solution consisting of sodium chloride, alcohol, glycerine, and a small amount of carbolic acid.

This extract was employed in the treatment of eighteen cases of typhoid, being given in from teaspoonful to tablespoonful doses every two hours, and in all cases marked improvement was noted; the pyrexia became remittent and soon disappeared, the pulse improved, diarrhœa ceased, and convalescence soon set in. No unpleasant secondary effects were observed.

In a subsequent communication² Jez again recommends this extract for further use in typhoid fever, and regards it as specific in action, harmless, and therefore capable of being used in large doses, while it is also useful in reaching a differential diagnosis. According to Jez, if used continuously in typhoid fever, it lowers the body temperature, strengthens the pulse, shortens the duration of the disease, and lessens or neutralizes the action of the typhoid toxins.

Markl³ found that the Jez extract possessed slight antibactericidal effects upon the typhoid bacilli, but was not antitoxic, while Walker⁴ obtained absolutely negative results with the Jez extract, but himself prepared an antityphoid serum, which he found to be both protective and curative.

¹ Wien. med. Wchnschr., Feb. 18, 1899.

² Ibid., 1901, No. 4.

³ Ibid., 1902, No. 3.

⁴ J. Path. and Bacteriol., Nov., 1901.

Mossé and Frenkel¹ report a case in which the agglutinating power of a woman with typhoid fever was shown to be transmitted to her child born during the fever. Their conclusions from a consideration of this and other cases are: (1) The typhoidal agglutinating property may pass from mother to fœtus through the non-altered placenta. (2) This property may be found in the newborn if the mother has had typhoid fever during the gestation. (3) The reaction is much less marked in the child than in the mother, and diminishes rapidly after birth. (4) The energy of the agglutinating power of the mother, and especially the length of time in which the agglutinating substances have been impregnating the placenta, are important conditions of the transmission of the agglutinating power from mother to child. (5) The agglutinating power of the mammary secretion, usually much less than that of the blood, may, in certain cases, reach a high degree (sometimes one to one hundred).

Delamarre and Chaillou have also reported a case of the same nature and arrive at the same conclusions.

Rouslacroix² reports some extremely interesting cases of the passage of the agglutinin from the mother to the fœtus during typhoid fever. From a consideration of his cases and the cases in the literature he concludes that the presence or absence of toxins, antitoxins, and agglutinins in the fœtal blood depends largely upon the length of time elapsing between the beginning of the maternal typhoid and the expulsion of the fœtus, for the placenta during the beginning of an attack of typhoid acts as a distinct barrier to their passage. Thus the agglutinins are more likely to be found in mild cases of typhoid fever, where the delivery occurs towards the end of the attack than in those intense infections of the mother which so often bring about an extremely rapid expulsion of the fœtus. The liver of the fœtus seems to furnish a second barrier to the general diffusion of the agglutinins in the fœtal organism.

(c) DIPHTHERIA.

The use of diphtheria antitoxin as a remedial agent is so firmly established that further discussion on the subject is absolutely

¹ Presse méd., Jan. 14, 1899.

² Ibid., Apr. 2, 1902.

unnecessary; suffice it to say that it is one of the most valuable therapeutic agents in medicine.

As to the duration of immunity in prophylactic injections, Jump,¹ from a consideration of the experiments of Behring, Rubens, Bornstein, and Bullock on animals, and Lehr, Rietter, Morrill, Biggs, and Guerard and Donald on human beings, concludes that immunity will last three weeks if a reliable antitoxin is used, and recommends that in the case of exposed individuals two hundred and fifty units be given to all under two years of age, five hundred units to all others.

As to the effect of serum therapy in diphtheritic laryngeal stenosis, Galatti,² from a careful analysis of his cases (twenty-nine before the use of antitoxin, thirty-two afterwards), concludes that the mortality has markedly diminished by its use (forty-seven and eight-tenths per cent. in the first series, five and one-half per cent. in the second).

Various interesting questions regarding the chemical changes in the blood after the use of diphtheria antitoxin have been discussed by Karfunkel.³ He especially considers the changes of the alkalinity of the blood after the administration of diphtheria toxin and antitoxin at normal and at artificially increased temperatures. The results of his experiments were as follows:

1. Gradual increase of the temperature did not change the alkalinity of the blood; sudden and marked increase of temperature lessened the alkalinity considerably.
2. After intravenous injections of one cubic centimetre of diphtheria antitoxin at room temperature there was after one to two hours a considerable decrease in the alkalinity. Gradual heating brought about a rapid decrease of the blood alkalinity and a fatal termination of the infection.
3. After intravenous injection of diphtheria antitoxin at normal body temperature a distinct increase of alkalinity is noted, while, under the influence of artificial, gradual increase of temperature, there is neither an increase of alkalinity nor any special harm to the blood.

¹ Phila. M. J., Jan. 11, 1902.

² Wien. med. Wehnschr., 1901, Nos. 2 and 3.

³ Ztschr. f. Hyg. u. Infektionskrankh., vol. xxxii. p. 149.

4. After intravenous injection of two parts of pure antitoxin and one of toxin, the alkalinity of the blood at room temperature is significantly decreased, while at artificially increased temperature the amount of alkali remains intact and the animals experimented on remain well.

(d) TUBERCULOSIS.

In tuberculosis an enormous amount of work has been done upon all phases of the subject of serum therapy, serum diagnosis, and immunity in this disease. In this article there will be time only briefly to mention a few of the more recent articles, discussing first serum diagnosis, then serum therapy and concluding with a few remarks on the diagnostic and therapeutic value of tuberculin.

Many members of the French school have described a typical agglutinating reaction in tuberculosis, but the recent work of the German and Italian schools is absolutely opposed to this.

De Grazia¹ finds that the cultures of tubercle bacilli are agglutinated not only by the serum of tuberculous individuals, but also by the serum of completely normal individuals, or by that of those suffering with other diseases.

Furthermore, De Grazia found that the serum of tuberculous individuals energetically agglutinated other bacteria, such as the diphtheria, typhoid, cholera, and colon bacilli, and the staphylococcus pyogenes aureus, and therefore denies the right of any one to speak of a specific agglutinating reaction in tuberculosis. He also found that the degree of the reaction bore absolutely no relation to the intensity or the extent of the infection.

Ruitinga² performed a series of experiments upon the serum diagnosis of tuberculosis, using from ten- to twelve-day old cultures of the bacillus, made from a stock culture furnished by Arloing. The examinations were made during a period of eight hours in dilutions of one to five, one to ten, and one to twenty.

The serum of sixty patients with or without demonstrable tuberculosis and of three normal and six tuberculous cows was tested. In the case of patients with pulmonary tuberculosis and with tubercle bacilli in their sputum there were four negative, nine positive results; the reaction was not obtained in lupus; the charac-

¹ Berl. klin. Wehnschr., 1902, Nos. 11 and 12.

² Dissertation, 1901.

ter of the reaction did not bear the slightest relation to the course or character of the disease, while in the non-tuberculous patients the reaction was positive eleven times, negative nine times. For these reasons, Ruitinga feels that the reaction cannot be recommended.

Beck and Rabinowitsch¹ give the results of their investigation of the blood of seventy-eight freshly slaughtered cattle, partly healthy, partly tuberculous, as regards the agglutinating properties of the serum. The method used was that originally devised by Arloing and Courmont, with which these investigators obtained such positive results.

Beck's and Rabinowitsch's results were absolutely opposed to those of the French investigators, for positive results were obtained in healthy animals, in tuberculous animals, and in those suffering from other diseases. This and the results of their earlier investigations upon human beings² make them conclude that the reaction is absolutely valueless in the diagnosis of tuberculosis.

Perhaps the most work upon serotherapy in tuberculosis has been done by Maragliano, who has been experimenting upon this subject for a number of years in both human beings and animals. In a recent article³ this observer gives the result of his recent work.

Maragliano furnishes his tuberculous antitoxin now in tubes holding one, five, and ten cubic centimetres respectively. In the slowly progressive cases with no or very slight fever he recommends one cubic centimetre every two days; if there is fever, he recommends the same method of treatment, but if after five doses the fever is not diminished, he gives five cubic centimetres every two days, and if again after five days there is no effect from the temperature, ten cubic centimetres are given every two days. If after the use of the antitoxin for a month the disease is not checked, its further use is valueless. Its first manifestations are in the favorable results it has upon the tuberculous toxæmia, dyspepsia, anæmia, night-sweats, and fever; later an increase in weight is noted, while still later the local condition in the lungs shows improvement.

From a long experience with his antitoxin the author concludes

¹ Deutsche med. Wehnschr., 1902, No. 10.

² Centralbl. f. innere Med., 1900, No. 32.

³ Gazz. d. osp., 1901, No. 151.

that if the symptoms are not influenced by the serum, that either a secondary infection has entered in, or that the diseased organism is not able to respond to the stimulus of the injected antitoxin. There are no contra-indications to its use, although occasionally erythema or urticaria follows the injection.

Figari and Lattes¹ give their results with the use of Maragliano's serum in one of the hospitals for consumptives. One hundred and seventy-one cases in all were treated between the 1st of January, 1900, and the 1st of June, 1901, and of these one hundred and seventy-one cases forty-four were completely cured, seventy-six were benefited, and thirty-nine remained stationary, while twelve became worse, although none died.

Nothing new is added by the authors as to the exact mode of action of the serum; according to them the toxicity of the blood is lessened, and the circulating toxins are neutralized, while the resistance of the body is increased, and thus we have a secondary bactericidal effect.

The diagnostic value of tuberculin is almost universally recognized, but only a few, including, of course, Koch himself, are making use of tuberculin therapeutically, and the trend of modern medical opinion is certainly against its use in this connection.

Goetsch² describes the treatment with tuberculin of pulmonary tuberculosis as practised by him under the advice of Koch during the past ten years. Of one hundred and seventy-five cases, one hundred and twenty-five were cured, while the other fifty were benefited, but were not cured. Perhaps these results are better understood when it is stated that only in forty per cent. of the cases tubercle bacilli were found. The method employed was to determine the maximum dose which would not be associated with an increase of temperature, and then to gradually increase the dose, carefully watching the temperature reaction.

Hoke,³ however, noted absolutely no improvement after the continued use of Ponzio's tuberculin in eight cases in Von Jaksch's clinic, all of which were most favorable prognostically, and concludes that it is not to be recommended in the treatment of tubercu-

¹ Gazz. d. osp., 1901, No. 117.

² Deutsche med. Wchnschr., 1900, vol. xxvi., No. 2.

³ Ztschr. f. Heilk., 1901, Nos. 8 and 9.

losis, a conclusion to which most experimenters along this line thoroughly agree.

(e) TETANUS.

After diphtheria tetanus has been the one disease in which investigators and clinicians have hoped and expected that successful results would be obtained by the antitoxin treatment. Unfortunately, however, the practical results have not borne out these hopes.

Behring¹ recommends the use of one hundred units (ten cubic centimetres) of his serum, the dose to be repeated the next day; he recommends the subcutaneous method of injection, and also lays stress upon the value of the application of the antitoxin to the infected wound. In another communication² he insists that the serum treatment, to offer any chance of success, must be inaugurated within thirty hours of the appearance of the first symptoms of tetanus.

Török,³ from the results obtained in an exhaustive series of experiments upon mice, rabbits, and guinea-pigs, concludes that "the antitoxin treatment is most efficacious," but unfortunately the practical results of the use of antitoxin in tetanus in man do not harmonize with the conclusions reached by Behring and Török.

Steuer,⁴ while of course admitting the immunizing power of the serum in those cases either exposed to infection, or after the infection has just started, concludes that its use in cases with well-marked symptoms is attended with no results, even if the injection be given within thirty hours of the first manifestations of the disease; for, in the vast majority of cases, tetanus is absolutely unsuspected until the appearance of the first symptoms, that is, until the patient is thoroughly saturated with the poison. This is borne out by the series collected by Lambert,⁵ by Toeper and Oppenheim,⁶ and by Möllers.⁷

¹ Therapie der Gegenwart, Mar., 1900.

² Deutsche med. Wehnschr., 1900, vol. xxvi., No. 2.

³ Ztschr. f. Heilk., 1900, vol. xxi., No. 36.

⁴ Centralbl. f. d. Grenzgeb. d. Med. u. Chir., 1900, vol. iii., Nos. 5-11.

⁵ Med. News, July 7, 1900.

⁶ Arch. gén. de Méd., 1900, vol. iii., No. 4.

⁷ Deutsche med. Wehnschr., 1901, No. 47.

Almost all investigators agree that the subcutaneous injection is preferable to the intracerebral and subdural methods.

Among other methods of treatment should be mentioned the subcutaneous injection of emulsion of brain-substance, and Wassermann, Krokiewicz, Zupnik, Baginski, and Mori and Salvolini agree that in their cases treated by this method the symptoms diminished in a marked degree after the injections.

(f) PNEUMONIA AND PNEUMOCOCCUS INFECTIONS.

In pneumonia, work has been carried on upon two distinct lines, one, the diagnosis of pneumonia by means of an agglutination reaction, the other, the application of serotherapy to pneumonia; we will also discuss briefly the diagnosis of pneumonia by means of cultures from the blood.

Huber¹ describes an extremely interesting method by means of which the agglutination test may be applied to pneumococcus infections; the test, however, is practically the same as that devised by Besançon and Griffon. Instead of the usual method of testing bouillon-cultures of the pneumococcus with the serum from the suspected patient, the procedure is to transplant the pneumococcus in tubes containing the patient's serum, carefully separated from the red blood-corpuscles by centrifugalization.

In normal human or animal serum the pneumococci at a temperature of from 35.5° C. to 37° C. develop as a diffuse clouding of the serum; but in the case of serum from a patient infected with the pneumococcus the serum remains perfectly clear, while on the bottom of the tube is seen a ball-like mass or a membrane made up of the agglutinated cocci; this mass may be so firm that even shaking will not break it up, while microscopically the mass presents the usual appearance of agglutinated bacteria. The reaction is most marked near the time of the crisis; the earliest day on which Huber obtained the reaction was the fifth, while after the crisis this agglutinating power rapidly disappeared, so that ten days after the crisis but a weak reaction was obtainable. Huber tested the reaction with serum from cases of acute articular rheumatism, tonsillitis, and ulcerative endocarditis, but always with negative

¹ Centralbl. f. innere Med., 1902, No. 17.

results, although, of course, a greater number of observations must be made before the specificity of the serum is proved.

While as a rule this serum reaction will not be necessary in pneumococcus pneumonia, still it will be of value in the masked forms of pneumonia, especially central pneumonia with typhoid symptoms, and further also in sepsis, endocarditis, meningitis and other diseases, which may be due to an infection with the diplococcus pneumoniæ.

This work is of great interest from the fact that it suggests again the possibility that we may obtain a healing serum in pneumonia, as in the majority of cases the formation of the immunizing substances runs parallel to that of the agglutinins.

Daddi and Pesci¹ give the results of their experiments in the agglutination of the diplococcus by the blood-serum. In thirty-five cases of lobar pneumonia the blood-serum agglutinated positively thirty-one times, and the reaction was absent four times; in these latter cases the serum would not even agglutinate the diplococcus obtained from the sputum of the affected individuals. In most of the cases the reaction appeared on the third or fourth day of the disease and lasted a week or less. In one case the reaction lasted thirty-five days after the crisis.

In various other infections due to the pneumococcus positive results were obtained, as various cases of pleurisy, nephritis, and tonsillitis, while agglutination never took place in healthy individuals or those suffering from other diseases.

Besides the diagnosis of pneumococcus infections by means of the agglutination reaction, various investigators have attempted to make this diagnosis by means of blood cultures.

Prochaska² made cultures in fifty cases of pneumonia, using a large quantity of blood in each case. In all cases micro-organisms were found; in the great majority of cases the pneumococcus was found in pure culture, twice the staphylococcus as well, and twice in place of the pneumococcus a peculiar streptococcus which perhaps is a form related to the pneumococcus. As a usual rule, cultures could be obtained by using a large quantity of blood, the transplantation taking place in bouillon.

¹ Rev. d. clin. méd., 1901, No. 21.

² Deutsches Arch. f. klin. Med., vol. lxx., Nos. 5 and 6.

Cole¹ has made careful blood-cultures, using large quantities of blood in thirty cases of pneumonia, and in nine of these cases pneumococci were obtained in the cultures. All these nine cases ended fatally, and Cole's results, therefore, do not agree with those of Prochaska, who found the pneumococci in all of the fifty cases examined by him, and who thought that the severity of the disease bore no relation to the finding of the cocci.

The result of serotherapy in pneumonia has up to the present time been distinctly disappointing, for, notwithstanding the positive results of the Klemperer brothers in successfully immunizing rabbits against pneumococcus infections by the injection of cultures of the micro-organism, or of serum or fluids of animals rendered immune, or of persons or animals after the crisis of pneumonia had been passed, nevertheless the results in the serum treatment of pneumonia in human beings has so far proven unsatisfactory. A number of members of the Italian school, as Pane, Maragliano, Cantieri, Caruso, and others have reported a number of cases of pneumonia successfully treated by this method, although the reports are too meagre in regard to details of importance to warrant any definite conclusions being drawn from them. Washburn, the Klemperers, Canby, Frey, and Everhart report a few successful cases, while Wiesbecher reports five cases successfully treated by serum obtained from other patients after the crisis. On the other hand, the majority of observers report practically negative results with the anti-pneumococcus serum in the treatment of pneumonia; thus, Banti and Pieraccini obtained absolutely negative results in twenty-one cases so treated, and Sears² reports twelve cases treated with anti-pneumococcus serum without being able to show any definite improvement, although his cases were distinctly unfavorable ones.

Yet while the results have so far proved discouraging, nevertheless the positive results of animal experiments, the apparent success of the treatment in a few cases, and the great need for a rational line of therapy in pneumonia should all tend to make experimenters and clinicians unite in testing any and all of the theoretically, at least, rational methods of treatment of this disease.

¹ Johns Hopkins Hosp. Bull., June, 1902.

² Boston M. and S. J., 1901.

(g) BUBONIC PLAGUE, CHOLERA, MALTA FEVER, LEPROSY, AND EPIDEMIC DYSENTERY.

In bubonic plague interesting work has been done during the past few years both in relation to serum diagnosis and serum therapy.

Cairns¹ has tested the agglutinating property of the blood-serum of twenty-five patients suffering with bubonic plague, making three hundred tests in all; emulsions of the bacilli in 0.75 per cent. sodium-chloride solution were used. In the early stages of the disease the reaction was often wanting, and thus was not observed in the cases which terminated fatally with great rapidity. The agglutinating tendency increased from the first to the sixth week, remained then constant until the eighth week, after which it gradually diminished; in some cases it was present five months after the disease.

Kolle and Martini,² in an article in which many interesting points in connection with plague are considered, discussed the curative property of the Yersin and Lustig serum, and also the agglutinating properties of the serum of infected animals and individuals. In regard to the first they have found that it exerts a slight prophylactic effect, and therefore recommend it to be used as a last resort; as regards the second, that agglutination often took place from one to six thousand.

Klein³ has employed a similar method in testing the agglutination of the plague bacillus in bubonic plague, using emulsions from the bacilli grown in gelatin in 0.75 per cent. sodium-chloride solution. He obtained positive results in the cases of patients suffering with the disease in dilutions of one to twenty and one to forty, and also in the case of infected rats, but not in the case of healthy mice and healthy men.

Zabolotny⁴ found in forty cases of plague that the serum reaction was absent in the first week, in the second week present in dilution of one to ten, and in the third or fourth week in dilution of one to fifty. Yersin reports very favorably results with the anti-

¹ *Lancet*, June 22, 1901.

² *Deutsche med. Wehnschr.*, 1902, Nos. 1 and 2.

³ *Lancet*, Feb. 16, 1901.

⁴ *Deutsche med. Wehnschr.*, 1897, p. 392.

plague serum, while Haffkine reports most favorable results from his protective inoculations in individuals exposed to the plague.

In cholera, Grüber, Durham, and Pfeiffer have shown that human cholera serum would clump cholera bacilli, and Achard and Bensaude have used this successfully in diagnosing cholera in man.

Haffkine has employed quite successfully in India preventive inoculations, while Blackstein, from the fact that a dilute solution of chrysoidin agglutinates the cholera bacteria and also has a bactericidal action, recommends dilute chrysoidin solutions as an internal disinfectant and preventive agent during epidemics of cholera.

In Malta fever a typical clump reaction of the micrococcus melitensis has been described by Wright and Smith in fifteen cases, by Curry in eighteen cases, by Strong, Cox, Musser, and others. The reaction was present often in dilutions of one to fifty and higher, while negative results were always obtained with typhoid bacilli.

In leprosy, Sprunk¹ has applied serum diagnosis; he has cultivated the bacillus of leprosy upon gelatinized horse's serum, and has found that the blood of patients suffering with leprosy agglutinates this bacillus in a characteristic way.

In the epidemic dysentery of the Orient, due to Shiga's bacillus, Shiga himself has experimented for the past three years upon various herbivora, especially horses and sheep, in the hope of obtaining an immunizing serum, this serum being prepared in the usual way. That this serum gives great promise of success may be seen from the reports of the cases in which it has been used, although its expense at present militates against the possibility of its general use. In 1900 Shiga reported two hundred and sixty-six cases treated with this serum, with a mortality of less than ten per cent., while the mortality in the same hospital during this period among cases not so treated was between thirty-five and thirty-seven per cent.

Most important contributions to the subject of serum diagnosis and serum therapy in dysentery are expected from Flexner and his students, who are devoting much time to this subject in the Pathological Laboratory of the University of Pennsylvania; and already Duvall and Bassett have practically proved that Shiga's bacillus is the cause of many cases, at least, of the summer gastro-enteritis of infants.

¹ Semaine méd., 1898, vol. xviii., No. 49.

(h) INFECTIONS WITH COLON, PYOCYANEUS AND PROTEUS BACILLI,
AND WITH THE STREPTOCOCCUS.

The agglutination reaction during the past few years has been applied in a small number of cases to a considerable variety of micro-organisms, among which we may mention the colon bacillus, the bacillus pyocyaneus, and the bacillus proteus. In the case of the colon bacillus considerable work has been done in Escherich's laboratory in connection with the rôle played by the colon bacillus in the diarrhœas of infancy. H. L. Smith, working in Escherich's laboratory, showed definitely that the colon bacillus causing the diarrhœa could be agglutinated by the serum of the infant from whose stools this especial bacillus had been grown, while it could not be agglutinated by the serum of other infants, notwithstanding the fact that they were suffering from diarrhœa at the same time.

Kreisel¹ has carried on further studies along these lines in the same laboratory, attempting also a series of animal experiments, and his results were entirely confirmatory of those obtained by Smith. The same results were obtained by Radziewsky,² who reaches practically the same conclusions.

T. R. Brown³ reports two cases of cystitis of long duration, one due to the colon bacillus, the other to the bacillus proteus vulgaris, in each of which positive agglutination reactions were obtained with the respective patient's serum in dilutions of one to fifty.

Leclainche and Moore⁴ have prepared a serum immunizing against the vibrio septicus, the serum being prepared by the intravenous injections of cultures of the micro-organism into various animals. This serum possesses protective properties and, under certain conditions, healing properties as well.

The serum is bactericidal as well as antitoxic, and its properties apparently depend upon the favorable action it exerts upon phagocytosis.

Although an occasional report is seen of a case in which the antistreptococcus serum has been used successfully, nevertheless the large majority of physicians who have used it have been unable to

¹ Centralbl. f. Bakteriöl., 1901, vol. xxix. p. 6.

² Ztschr. f. Hyg. u. Infektionskrankh., 1900, vol. xxxiv. p. 369.

³ Boston M. and S. J., Nov. 8, 1900.

⁴ Ann. de l'Inst. Pasteur, 1901, vol. xv. p. 1.

detect any beneficial effects from its use in streptococcus infections in human beings, although Marmorek's experiments upon animals gave promise of success.

Scharfe¹ has employed Marmorek's antistreptococcus serum in twenty-three cases of puerperal infection. Scharfe concludes that this treatment has absolutely no influence upon the course of the infection, nor is it useful diagnostically, but in some cases it seems to possess some prognostic value, for cases in which the pulse and temperature fell from twenty-four to thirty-six hours after the injection usually terminated favorably.

(i) PERTUSSIS.

Silvestri² has used the blood-serum of convalescents from whooping-cough in the treatment of this disease. It was used in seven cases, fifteen to twenty cubic centimetres of the serum being injected in each. In all cases the effect was strikingly favorable; in two cases, however, the injection had to be repeated.

(j) OTHER WORK IN SERUM THERAPY AND IMMUNITY.

Vaerst³ has experimented upon the immunizing action of pyocyanese against anthrax at the same time as similar experiments were being tried by Emmerich and Löw.

Pyocyanese is obtained by destroying six-weeks-old bouillon cultures of the bacillus pyocyanus by exposing them to a temperature of 58° C. and precipitating with ammonium sulphate.

Pyocyanese possesses not only a preventive action upon the development of anthrax, but also a bacteriolytic action upon the microbe itself. The injection of pyocyanese made at the same time as that of the anthrax bacilli prevents the development of anthrax in the animal experimented upon, but the repeated preventive inoculation of a watery solution of pyocyanese does not immunize rabbits against the subsequent inoculation with the anthrax bacillus. The serum of animals inoculated with pyocyanese protects other animals against the fatal dose of the bacteria, while the extracts of the spleen of animals treated with pyocyanese does not protect other animals against anthrax.

¹ Beitr. z. Geburtsh. u. Gynaek., 1900, vol. iii. p. 226.

² Gazz. d. osp., 1901, No. 114.

³ Centralbl. f. Bakteriöl., vol. xxxi. p. 293.

The question of immunity against malaria has been much discussed since Koch's expedition to Africa, which led him to believe that there is both a natural and an acquired immunity against malaria. While this view is held by a few observers, especially the Germans, the great majority of physicians and investigators living in malarial countries hold the opposite view.

Plehn¹ gives the results of his studies in Cameroon, where he has been the physician appointed by the German government to pursue such studies, and according to him most of the inhabitants of equatorial West Africa possess a relative, many an absolute immunity. Also in the case of Europeans who remain in West Africa, a relative immunity is established by the constant use of quinine.

In a most carefully prepared article Glogner² opposes Koch's views regarding the inheritance or the acquisition of immunity in malaria, and his figures, which were obtained from the study of a large number of cases of malaria among soldiers, children, and natives in Java, all go to show that there is no inherited or acquired immunity in this disease. Thus the mortality among the native soldiers is the same as that among the European soldiers, and everywhere in the Malayan Archipelago are found evidences of malaria, which is difficult to explain on Koch's theories. Also in the Protestant Orphan Asylum at Samarang, of the children between seven and thirteen years of age, ninety-two and three-tenths per cent. had malaria, and thirty-three per cent. anæmia, while of those between thirteen and twenty-one years of age ninety-one and seven-tenths per cent. had malaria, and thirty-seven and one-half per cent. had anæmia, that is, there was practically no difference in the prevalence between the older and the younger children.

Glogner believes that the examination should not be confined to a study of the blood findings alone, but that the presence of an anæmia and of an enlarged spleen should also be taken into consideration.

No article on serum therapy could be complete without a reference to the serum treatment of snake-poisoning. The work of Calmette upon animals which have been experimentally inoculated with snake-venom, and in the case of human beings who have been

¹ Jena, G. Fischer, 1901.

² Virchow's Arch., vol. clxii., No. 2.

accidentally bitten, shows that much is to be expected from the serum treatment of snake-poisoning. In one of his articles Calmette concludes as follows: "Animals may be immunized against the venom of serpents either by means of repeated injections of doses, at first feeble, and becoming progressively stronger, or by means of successive injections of venom mixed with certain chemical substances, among which I may especially mention the chloride of gold and the hypochlorides of lime and soda; the serum of animals thus treated is at the same time preventive, antitoxic, and therapeutic, exactly as is that of animals immunized against diphtheria and tetanus."

Any one especially interested in the subject of the relation of snake-venom to hæmolysis, bacteriolysis, and toxicity is referred to the article of Flexner and Noguchi¹ on this subject.

¹ J. Exper. Med., 1902, vol. vi., No. 3, p. 277.

INDEX TO VOLUME IV.

(TWELFTH SERIES.)

A

Abdominal diagnosis, 63
 Abscesses, the blood in, 260
 Addison's disease, the blood in, 274
 Altitudes, high, influence of, upon the constitution of the blood, 228
 Amblyopia, crossed, 120
 Anæmia, pernicious, 231
 with extensive pigmentary changes in the skin, 47
 splenic, 241
 treatment of, 242
 Anæmias, the, 229
 secondary, 235
 Anæsthesia, general, preliminary suggestions in, 158
 Anatomy of the inguinal region, 136
 Anchylostomiasis, the blood in, 259
 Aneurisms of the aorta, detection of, 88
 situations of, 87
 some clinical aspects of, 81
 treatment of, by gelatin in hypodermic injections, 86
 Anorexia, 46
 Aorta, aneurisms of the, 81
 Appendicitis, the blood in, 260
 Artery, common carotid, ligature of the, 175
 posterior tibial, ligature of the, 176
 Asthma, 41
 bronchial, the blood in, 274

B

Bacteriological study of the blood, 216
 Bassini operation, 137
 Beriberi, the blood in, 270
 Binocular diplopia, clinical significance of, 188
 Bishop, E. Stanmore, abdominal diagnosis, 63
 Blood at different periods of life, 225
 bacteriological study of the, 216
 constituents of, 206
 examination, methods of, 210
 fluid portion of, 207
 human, differentiation of, from that of animals, 228
 in abscess, 260
 in acute articular rheumatism, 256
 in Addison's disease, 274
 in anchylostomiasis, 259

Blood in appendicitis, 260
 in beriberi, 270
 in bronchial asthma, 274
 in cancer and diseases in the gastro-intestinal tract, 267
 in cardiovascular diseases, 264
 in chicken-pox, 255
 in chorea, 269
 in convulsions, 270
 in diabetes mellitus, 266
 in diphtheria, 249
 in diseases due to animal parasites, 257
 to convulsions, 270
 to intestinal parasites, 258
 of nutrition and metabolism, 266
 of the gastro-intestinal tract, 267
 of the internal genital organs in women, 273
 of the skin, 271
 in epidemic cerebrospinal meningitis, 255
 in exposure to cold and heat, 227
 in filariasis, 258
 in functional neuroses, 269
 in general paralysis, 269
 in gonorrhœa, 254
 in gout, 266
 in health and in disease, with a review of the recent important work on this subject, 205
 in high altitudes, 228
 in infectious diseases, 248
 in inflammatory processes, 260
 in influenza, 254
 in jaundice, 274
 in labor, 271
 in lead poisoning, 272
 in malaria, 257
 in Malta fever, 254
 in measles and German measles, 252
 in mental and nervous diseases, 269
 in nephritis, 264
 in other infectious diseases, 256
 in other intestinal parasites, 260
 in pertussis, 249
 in plague, cholera, and yellow fever, 254
 in pneumonia, 258
 in poisoning with coal-tar preparations, 272
 in pregnancy, 271

- Blood in puerperium, 271
 in purpura, 267
 in scarlet fever, 252
 in smallpox, 255
 in special diseases and pathological conditions, 248
 in syphilis, 252
 in thyroidectomy, 274
 in tonsillitis, 256
 in trichinosis, 258
 in tuberculosis, 248
 in typhoid fever, 250
 in various intoxications, 272
 physical and chemical examination of, 216
 specimens, dried and stained, examination of, 211
 fresh, examination of, 210
 Blood-cells, red, and granulations in the red blood-cell, 219
 various, the origin and significance of, 219
 white, and granulations in the white blood-cells, 222
 Blood-corpuscles, red, 207
 estimation of the, 214
 white, 209
 estimation of the, 215
 Blood-granulations, origin and significance of, 219
 Blood-plates, estimation of, 215
 Bodine, J. A., fright and deaths in chloroform narcosis; mental preoccupation as a preliminary to general anesthesia; resection of tuberculous testicle, 153
 Bosanquet, William Cecil, the nature of cancer and other new growths, 77
 Brain, traumatic lesions of, in their relation to operation, 102
 Bromides, triple, 43
 Bronchiectasis, 41
 Bronchitis, chronic, 40
 Brown, Thomas R., the blood in health and in disease, with a review of the recent important work on this subject, 205
 Bubonic plague, serum diagnosis of, serum therapy in, and immunity from, 295
- C**
- Cancer, the blood in, 267
 and other new growths, the nature of, 77
 Cardiovascular diseases, the blood in, 264
 Carotid artery, common, ligature of the, 175
 Catarrh, chronic gastric, treatment of, 7
 Cerebrospinal meningitis, epidemic, the blood in, 255
 Chase, Robert H., some aspects of parana, 97
- Chicken-pox, the blood in, 255
 Chloroform narcosis, fright and deaths in, 153
 Chlorosis, 233
 Cholera, the blood in, 254
 serum diagnosis of, serum therapy in, and immunity from, 296
 Chorea, the blood in, 269
 Coagulation-time, estimation of, 215
 Coal-tar preparations, poisoning with, the blood in, 272
 Cold, influence of exposure to, upon the constitution of the blood, 227
 Colds, flaxseed tea for, 40
 Colon bacilli, infections with, serum diagnosis of, serum therapy in, and immunity from, 297
 Convulsions, diseases associated with, the blood in, 270
 Cough mixtures, 39
 Crossed amblyopia, 120
- D**
- Diabetes mellitus, the blood in, 266
 Diagnosis, abdominal, 63
 and treatment, early, of malignant disease of the larynx, 28
 Diarrhoea from cold, 42
 Diphtheria, serum therapy in, 287
 the blood in, 249
 Diplopia, binocular, the clinical significance of, 188
 Dysentery, epidemic, serum diagnosis of, serum therapy in, immunity from, 296
- E**
- Effusion, purulent pericardial, in pneumonia, 51
 Elsendrath, Daniel N., a case of meloplasty; tuberculous glands of the neck, 164
 Eruption, phlyctenular, recurring, of the fingers, 180
 Eye, motor anomalies of the, table of, 191
- F**
- Fallon, M. F., anatomy of the inguinal region and the radical cure of inguinal hernia, 136
 Filariasis, the blood in, 258
 Flaxseed tea for colds, 40
 Frontal sinus, opening of the, 177
- G**
- Gardiner, Charles Fox, sanatory tent and its use in the treatment of pulmonary tuberculosis, 1
 Gastric catarrh, chronic, treatment of, 7
 ulcer, hæmatemeses from, surgical treatment of, 144

Gastro-intestinal tract, the blood in diseases of, 267
Gelatin, treatment of aneurisms by hypodermic injections of, 86
Genital organs, internal, in women, the blood in diseases of the, 273
Glands, tuberculous, of the neck, 186
Glossolabial hemispasm, 117
Gonorrhœa, the blood in, 254
Gout, the blood in, 266
Growths, the nature of new, 77

H

Hæmatemesis from gastric ulcer, surgical treatment of, 144
Hæmoglobin, estimation of, 214
Hall, J. N., scars and marks of clinical interest, 71
Headache, powders, 43
Heart and lungs from a case of left-sided pneumonia with purulent pericardial effusion, 51
 massage and artificial respiration in narcosis, 174
Hemiplegia, functional and organic, diagnosis of, 112
 syphilitic, 122
Henry, Frederick P., lungs and heart from a case of left-sided pneumonia with purulent pericardial effusion, 51
Hernia, inguinal, radical cure of, 136
 suture material in, 140
Hinsdale, Guy, biographical sketches of eminent physicians, 198
Hysterical mutism, 133

I

Immunity, 275
Infectious diseases, the blood in, 248
Inflammatory processes, the blood in, 260
Influenza, the blood in, 254
Inguinal hernia, radical cure of, 136
 region, anatomy of the, 136
Intestinal parasites, diseases due to, the blood in, 258
Intestine, resection of the, 178
Intoxications, various, the blood in, 272

J

Jaundice, the blood in, 274

K

Keen, William W., biographical sketch of, 202
Kelly, Aloysius O. J., some clinical aspects of aneurisms of the aorta, 81

L

Labor, the blood in, 271
Lancereaux, E., treatment of aneurisms by gelatin in hypodermic injections, 36

Larynx, malignant disease of, early diagnosis and treatment of, 28
Lead palsy and trauma, 126
 poisoning, the blood in, 272
Lemonade, nutritive, 45
Leprosy, serum diagnosis of, serum therapy in, and immunity from, 296
Leser, Edmund, artificial respiration and heart massage in narcosis; Volkmann's suspension apparatus; ligature of the common carotid artery; opening of the frontal sinus; resection of the superior and inferior orbital nerves; suturing of the bowel, 174
Lesions, traumatic, of the brain in their relation to operation, 102
Leucocytosis, 243
 neutrophiles, 245
 pathological, 244
 physiological, 243
 polymorphonuclear eosinophilic, 246
Leucopenia, 247
Leukæmia, 236
Ligature of the common carotid artery, 175
 of the posterior tibial artery, 176
Lungs and heart from a case of left-sided pneumonia with purulent pericardial effusion, 51
 tuberculosis of the, and diseases which resemble it, 56
Lymphocytosis, 247

M

Malaria, the blood in, 257
Malta fever, the blood in, 254
 serum diagnosis of, serum therapy in, and immunity from, 296
Marks and scars of clinical interest, 71
Massage, heart, and artificial respiration in narcosis, 174
Measles, the blood in, 252
Meloplasty, 164
Mental preoccupation as a preliminary to general anæsthesia, 154
Motor anomalies of the eye, table of, 191
Moynihan, Berkeley G. A., the surgical treatment of hæmatemesis from gastric ulcer, 144
Mutism, hysterical, 133

N

Narcosis, artificial respiration and heart massage in, 174
 chloroform, fright and deaths in, 153
Neck, tuberculous glands of the, 186
Nephritis, the blood in, 264
Nerves, superior and inferior orbital, resection of, 178
Neuroses, the functional, the blood in, 269

O

- Opening of the frontal sinus, 177
Orbital nerves, superior and inferior, resection of the, 178

P

- Packard, the late Frederick A., pernicious anæmia with extensive pigmentary changes in the skin, 47
Palsy, lead, and trauma, 126
Paralysis, anomalous general, 131
 general, the blood in, 269
Paranoia, some aspects of, 97
Parasites, diseases due to, the blood in, 258
Pertussis, the blood in, 249
 serum diagnosis of, serum therapy in, and immunity from, 298
Phlyctenular eruption, recurring, of the fingers, with changes in the nails, possibly of hysterical origin, 180
Pill, tonic, 44
Plague, the blood in, 254
Plasma of the blood, 207
Pneumonia and pneumococcus infections, serum diagnosis of, serum therapy in, and immunity from, 292
 left-sided, lungs and heart from a case of, 51
 the blood in, 253
Poisoning, lead, the blood in, 272
 with coal-tar preparations, the blood in, 272
Powders, headache, 43
Pregnancy, the blood in, 271
Prescriptions, selected, 39
Pseudoleukæmia, 241
Puerperium, the blood in, 271
Purpura, the blood in, 267
Pyocyaneus and proteus bacilli, serum diagnosis of, serum therapy in, and immunity from, 297

R

- Resection of intestine, 178
 of superior and inferior orbital nerves, 178
 of tuberculous testicle, 161
Respiration, artificial, and heart massage in narcosis, 174
Rheumatic discomfort in the old, 44
Rheumatism, acute articular, the blood in, 256
 chronic, 42

S

- Santi, Philip R. W. de, some practical points on the early diagnosis and treatment of malignant disease of the larynx, 28

- Scarlet fever, the blood in, 252
Scars and marks of clinical interest, 71
Schlapp, Max, syphilitic hemiplegia; lead palsy and trauma; anomalous general paralysis; hysterical mutism, 122
Sedative, uterine, 43
Serum diagnosis, 275
 serum therapy, and immunity; infections with colon, pyocyaneus, and proteus bacilli, and with the streptococcus, 297
 of bubonic plague, cholera, Malta fever, leprosy, and epidemic dysentery, 295
 of diphtheria, 287
 of pertussis, 298
 of pneumonia and pneumococcus infections, 292
 of tetanus, 291
 of tuberculosis, 288
 of typhoid fever, 279
 reaction, 216
 therapy, 275
 and immunity, 298
Shoemaker, William T., the clinical significance of binocular diplopia, 188
Sinus, frontal, opening of the, 177
Skin, the blood in diseases of the, 271
Smallpox, the blood in, 255
Spiller, William G., traumatic lesions of the brain in their relation to operation, 102
Stern, Heinrich, treatment of chronic gastric catarrh, 7
Stewart, Purves, the diagnosis of functional and organic hemiplegia, 112
Streptococcus, serum diagnosis of, serum therapy in, and immunity from, 297
Suspension apparatus, Volkmann's, 175
Suture material in hernia operations, 140
Sweating, excessive, of the feet, 45
Syphilis, the blood in, 252
Syphilitic hemiplegia, 122

T

- Tent, sanatory, for the treatment of pulmonary tuberculosis, 1
Testicle, tuberculous, resection of, 161
Tetanus, serum diagnosis of, serum therapy in, and immunity from, 291
Thyroidectomy, the blood in, 274
Tibial artery, posterior, ligature of the, 176
Tonsillitis, the blood in, 256
Trauma and lead palsy, 126
Traumatic lesions of the brain in their relation to operation, 102
Treatment of aneurisms by gelatin in hypodermic injections, 36

Treatment of anomalous general paralysis, 133
 of chronic gastric catarrh, 7
 of hysterical mutism, 135
 of lead palsy, 129
 of malignant disease of the larynx, 28
 of pulmonary tuberculosis in sanatory tent, 1
 of syphilitic hemiplegia, 125
 surgical, of hæmatemesis from gastric ulcer, 144
 Trichinosis, the blood in, 258
 Tuberculosis, the blood in, 248
 of the lungs and diseases which resemble it, 56
 pulmonary, the sanatory tent in, 1
 serum therapy in, 288
 Tuberculous glands of the neck, 166
 testicle, resection of, 161
 Typhoid fever, the blood in, 250
 diagnosis of, 279
 treatment of, by serum therapy, 279

U

Ulcer, gastric, hæmatemesis from, surgical treatment of, 144
 Uncinariosis, the blood in, 259
 Uterine sedative, 43

V

Vaccination, 279
 Van Harlingen, Arthur, recurring phlyctenular eruption of the fingers, with changes in the nails, possibly of hysterical origin, 180
 Volkmann's suspension apparatus, 175

W

Whooping-cough, 44
 Wood, Horatio C., biographical sketch of, 198

Y

Yellow fever, the blood in, 254

GENERAL INDEX.

(TWELFTH SERIES.)

Volumes are indicated by Roman numerals: April, i.; July, ii.; October, iii.; January, iv.

A

Abdominal diagnosis, iv. 63
 tumor, iii. 206
 Abscesses, the blood in, iv. 260
 Acetopyrin, i. 252
 Acne, diagnosis of, i. 56
 rosacea, ii. 78
 treatment of, i. 54
 Acoln, i. 253
 Acopyrin, i. 253
 Actol, i. 253
 Addison's disease, the blood in, iv. 274
 Adrenalin in morphine and opium poisoning, i. 232
 Aërophagy, i. 246
 Airol, i. 253
 Akouphone, i. 294
 Albargin, i. 253
 Albuminuria, ii. 151
 Alcohol, wood, blindness from, i. 248
 Altitudes, high, influence of, upon the blood, iv. 228
 Amblyopia, crossed, iv. 120
 Anæmia, pernicious, iv. 47, 231
 splenic, iv. 241
 treatment of, iv. 242
 Anæmias, iv. 229
 secondary, iv. 235
 Anæsthesia, general, i. 256
 spinal, i. 259
 Anal fissure, i. 173; iii. 177
 Anastomosis, end-to-end, ii. 243
 Anchylostomiasis, the blood in, iv. 259
 Aneurism, aortic, i. 81, 236
 Aneurisms, iv. 36
 Angina, iv. 76
 Aniline oil, poisoning by, i. 248
 Anorexia, iv. 46
 Aorta, aneurisms of, iv. 81
 Aortic aneurism, treatment of, i. 236
 Appendicectomy, ii. 236
 Appendicitis, acute, ii. 213
 due to athletics, i. 262
 the blood in, iv. 260
 with ulcerative cæcitis, ii. 220
 Appendix, calculus in, i. 148
 Argentamin, i. 253
 Argonin, i. 253

Arsenical neuritis, i. 233
 Artery, common carotid, ligature of, iv. 175
 posterior tibial, ligature of, iv. 176
 Arthritis of the elbow-joint, ii. 221
 Arthrotomy, anterior, ii. 222
 Asphyxiation in delivery, i. 273
 Aspirin, i. 253
 Asterol, i. 253
 Asthma, iv. 41
 bronchial, the blood in, iv. 274
 conjunctival, iii. 64
 Astragalectomy for relapsed and inveterate club-foot, iii. 201
 Athletics and appendicitis, i. 262
 Autoinfection versus autointoxication, i. 118
 Autoinoculation of cancer, i. 280
 Autointoxication, autoinfection versus, i. 118
 definition of, i. 112
 gastro-intestinal, i. 107; ii. 155
 relation of thyroid gland to, ii. 162

B

Babcock, Robert H., pleurisy with more or less permanent pneumonic induration, —are they tuberculous? i. 102
 Bacteria, dissemination of, by ordinary air currents, i. 282
 Bacteriological study of the blood, iv. 216
 Baldy, J. Montgomery, abdominal tumor, iii. 206
 Basophilic granules in red corpuscles, i. 69
 Bassini operation, iv. 137
 Benedict, A. L., hepatic sclerosis; auscultatory percussion, ii. 102
 Beriberi, i. 269
 the blood in, iv. 270
 Beta-oxybutyric acid in the urine of diabetics, ii. 128
 Bile, iii. 258
 Binocular diplopia, iv. 188
 Biographical sketches of eminent living physicians, i. 1; ii. 247; iv. 198
 Bishop, E. Stanmore, abdominal diagnosis, iv. 63
 Bismal, i. 253

- Bismuthose, i. 253
 Black eye, ii. 79
 Bladder and rectal troubles in nervous diseases, ii. 15
 Blindness from wood alcohol, i. 248
 Bliss, Arthur Ames, acute mastoiditis; its dangers, its irregularity in symptoms, and the question of trephining, iii. 233
 Blood, at different periods of life, iv. 225
 bacteriological study of the, iv. 216
 constituents of, iv. 206
 effect of drugs upon, i. 40
 examination, iv. 210
 fluid portion of, iv. 207
 human, differentiation of, from that of animals, iv. 228
 in abscess, iv. 260
 in acute articular rheumatism, iv. 250
 in Addison's disease, iv. 274
 in anchylostomiasis, iv. 259
 in appendicitis, iv. 260
 in beriberi, iv. 270
 in bronchial asthma, iv. 274
 in cancer and diseases in the gastro-intestinal tract, iv. 267
 in cardiovascular diseases, iv. 264
 in chicken-pox, iv. 255
 in chorea, iv. 269
 in convulsions, iv. 270
 in diabetes mellitus, iv. 266
 in diphtheria, iv. 249
 in diseases due to animal parasites, iv. 257
 to convulsions, iv. 270
 to intestinal parasites, iv. 258
 of nutrition and metabolism, iv. 266
 of the gastro-intestinal tract, iv. 267
 of the internal genital organs in women, iv. 273
 of the skin, iv. 271
 in epidemic cerebrospinal meningitis, iv. 255
 in exposure to cold, iv. 227
 in filariasis, iv. 238
 in functional neuroses, iv. 269
 in general paralysis, iv. 269
 in gonorrhœa, iv. 254
 in gout, iv. 266
 in health and in disease, iv. 205
 in high altitudes, iv. 228
 in infectious diseases, iv. 248
 in inflammatory processes, iv. 260
 in influenza, iv. 254
 in jaundice, iv. 274
 in labor, iv. 271
 in lead poisoning, iv. 272
 in malaria, iv. 257
 in Malta fever, iv. 254
 in measles and in German measles, iv. 252
 Blood in mental and nervous diseases, iv. 269
 in other infectious diseases, iv. 256
 in other intestinal parasites, iv. 260
 in pertussis, iv. 249
 in plague, cholera, and yellow fever, iv. 254
 in pneumonia, iv. 253
 in poisoning with coal-tar preparations, iv. 272
 in pregnancy, iv. 271
 in puerperium, iv. 271
 in purpura, iv. 267
 in scarlet fever, iv. 252
 in smallpox, iv. 255
 in special diseases and pathological conditions, iv. 248
 in syphilis, iv. 252
 in thyroidectomy, iv. 274
 in tonsillitis, iv. 256
 in trichinosis, iv. 258
 in tuberculosis, iv. 248
 in typhoid fever, iv. 250
 in various intoxications, iv. 272
 physical and chemical examination of, iv. 216
 specimens, iv. 210
 Blood-cells, red, iv. 219
 various, iv. 219
 white, iv. 222
 Blood-corpuscles, red, i. 69; iv. 207
 white, iv. 209
 Blood-granulations, iv. 219
 Blood-plates, iv. 215
 Blood pressure in health and disease, i. 248
 Blood-test, i. 281
 Boas, I., habitual constipation, i. 21
 Bodine, J. A., fright and deaths in chloroform narcosis; mental preoccupation as a preliminary to general anæsthesia; resection of tuberculous testicle, iv. 153
 Boggess, Walter F., infantile spinal paralysis, iii. 142
 Boissard, A., the contest between the advocates of symphysectomy and the partisans of Cæsarean section, i. 187
 Borissosof, Peter, the function of the digestive glands, ii. 274; iii. 247
 Bosanquet, William Cecil, the nature of cancer and other new growths, iv. 77
 Bot-flies and their larvæ, ii. 108
 Bot-fly, the horse, iii. 114
 Bradycardia, iv. 148
 Bright's disease, i. 262
 Brinton, John H., remarks on some effects of fire-arms at short range, with experimental illustrations, iii. 148
 Bromides, triple, iv. 43
 Bromine, emulsions of, for epilepsy, ii. 77
 Brominol, i. 253
 Bronchiectasis, iv. 41
 surgical relief for, iii. 65

Bronchitis, chronic, iv. 40
 in the old, ii. 74
 morning appetizer in, ii. 74
 viscid, secretion in, ii. 75
 Brower, Daniel R., traumatic epilepsy;
 acute mania following injury; syringo-
 myella; cerebral hemorrhage, iii. 130
 Brown, Thomas R., the blood in health
 and in disease, with a review of the re-
 cent important work on this subject, iv.
 205
 Bubonic plague, serum diagnosis of, serum
 therapy in, and immunity from, iv. 296
 Bullet wounds, ii. 203

C

Cæcitis, ulcerative, appendicitis with, ii.
 220
 Cæsarean section, i. 187
 Calcium iodate, i. 253
 Calculus in appendix mistaken for cal-
 culus in the ureter, i. 148
 vesical, ii. 226
 Calmette's antivenine, cobra poisoning
 treated with, i. 249
 Cancer, i. 278; ii. 243
 alleged increase in the frequency of,
 i. 281
 autoinoculation of, i. 280
 inoperable, treatment of, i. 278
 the blood in, iv. 267
 the nature of, iv. 77
 Carcinoma, i. 278
 resection of sigmoid for, ii. 243
 Cardarelli, Antonio, surgical intervention
 in cases of great dilatation of the stom-
 ach, iii. 187
 Cardiovascular diseases, the blood in, iv.
 264
 Carotid artery, common, ligature of, iv.
 175
 Cataract, removal of, iii. 229
 Catarrh, chronic gastric, iv. 7
 Cauterization by hot air, iii. 65
 Cerebral hemorrhage, iii. 134
 thrombosis, bilateral, iii. 123
 Cerebrospinal fluid in cholæmia, i. 261
 meningitis, the blood in, iv. 255
 treatment of, by lumbar punc-
 ture, i. 261
 Cervical sympathetic, resection of, ii. 177
 Chappa, i. 281
 Chase, Robert H., some aspects of para-
 nola, iv. 97
 Chicken-pox, the blood in, iv. 255
 Child mortality in Chicago, i. 229
 Children, dosage for, i. 257
 Chirol, i. 253
 Chloralose, i. 253
 Chloretone, i. 253
 in epilepsy, i. 251
 Chloride of ethyl, i. 258

Chloroform narcosis, fright and deaths in,
 iv. 153
 Chlorosis, iv. 233
 Cholæmia, cerebrospinal fluid in, i. 261
 Cholera, the blood in, iv. 254
 serum diagnosis of, serum therapy in,
 and immunity from, iv. 296
 Chorea, the blood in, iv. 269
 Cirrhosis of the liver, i. 266; ii. 92
 Citric acid for ozæna, i. 64
 Clamp and cautery operation, iii. 163
 Club-foot, iv. 201
 Coagulation-time, estimation of, iv. 215
 Coal-tar naphtha, poisoning by, i. 249
 preparations, poisoning with, the
 blood in, iv. 272
 Cobra poisoning treated with Calmette's
 antivenine, i. 249
 Cocaine, spinal injection of, ii. 67
 Cold, influence of exposure to, upon the
 blood, iv. 227
 Colds, ii. 75; iv. 40
 Coley, William B., radical cure of inguinal
 and femoral hernia; sliding hernia;
 hydrocele of the canal of Nuck; epithe-
 lioma of the face; sarcoma of the
 upper jaw, ii. 189
 Colon bacilli, infections with, serum diag-
 nosis of, serum therapy in, and immu-
 nity from, iv. 297
 Conjunctival asthma, iii. 64
 Constipation, habitual, i. 21
 Convulsions, the blood in, iv. 270
 Cough mixtures, iv. 39
 Coxa vara, i. 127
 Crossed amblyopia, iv. 120
 Crothers, T. D., the treatment of mor-
 phinism, iii. 24
 Cuprol, i. 254
 Cystic fluids of the abdomen, trimanual
 method of percussion for detection of,
 i. 276
 Cystocele, radical cure of, i. 278
 Cysts, pancreatic, ii. 113

D

Daniel, P. L., pancreatic cysts, ii. 113
 Deafness, treatment of, by massage, iii. 56
 Debove, G. M., treatment of dilatation of
 the stomach by gastro-enterostomy, iii.
 180
 Decapsulization of the kidney, i. 262
 Dermoid cyst, removal of, i. 170
 Diabetes mellitus, ii. 1
 the blood in, iv. 266
 Diabetics, beta-oxybutyric acid in the
 urine of, ii. 128
 Diagnosis, abdominal, iv. 63
 and treatment of malignant diseases,
 iv. 28
 of osteomyelitis, iii. 36
 Diagnostic methods, newer, iii. 81

Diarrhœa from cold, iv. 42
 in the tuberculous, ii. 73
 Dieulafoy, G., means of telling whether an
 attack of serofibrinous pleurisy is tuber-
 culous, iii. 87
 Digestive glands, ii. 274; iii. 247
 Dilatation of the stomach, i. 96
 Dionin, i. 254
 Diphtheria, i. 214
 bacteriology and pathology of, i. 286
 serum therapy in, iv. 287
 the blood in, iv. 249
 Diplopia, binocular, iv. 188
 Diplogonoplos grandis, i. 283
 Dislocations and fractures and suits for
 malpractice, iii. 153
 Dormiol, i. 254
 Douglas, Carstairs, the presence and sig-
 nificance of beta-oxybutyric acid in the
 urine of diabetics and its relation to
 the coma, ii. 128
 Drainage, abdominal, i. 274
 suprapubic, ii. 248
 Drugs, action of, methods of investigating
 the, i. 37
 effect of, upon the blood, i. 40
 upon the nervous system, i. 41
 Drunkards' deaths resulting from pachy-
 meningitis hæmorrhagica, ii. 81
 Dymal, i. 254
 Dysentery, epidemic, serum diagnosis of,
 serum therapy in, immunity from, iv.
 296

E

Ectopia testis, crural, i. 132
 perineal, i. 131
 Edwards, Arthur R., classification of cir-
 rhosis of the liver, ii. 92
 Effusion, purulent pericardial, iv. 51
 Elsendrath, Daniel N., a case of melo-
 plasty; tuberculous glands of the neck,
 iv. 164
 Elbow, traumatism of, i. 176
 Electricity and silver wire in the treat-
 ment of aortic aneurism, i. 236
 Enteroptosis, i. 272
 Epicarlin, i. 254
 Epilepsy, chloretone in, i. 251
 emulsions of bromine for, ii. 77
 focal and Jacksonian, topical treat-
 ment of, i. 267
 traumatic, iii. 130
 Epithelioma of the face, ii. 200
 Eruption, phlyctenular, recurring, of the
 fingers, iv. 180
 Ethyl bromide and chloroform, i. 258
 Euguform, i. 254
 Eunatrol, i. 254
 Eupyrin, i. 252
 Exophthalmic goitre, enucleation of the
 thyroid for, i. 141

Eye, black, ii. 79
 motor anomalies of the, table of, iv.
 191
 syphilis of, iii. 218
 Eye-wash, ii. 79

F

Face presentation, iii. 213
 Fallon, M. F., anatomy of the inguinal
 region and the radical cure of inguinal
 hernia, iv. 136
 Fango, i. 254
 Fat necrosis, i. 273
 Faucial tonsils, methods of removal, iii.
 240
 Femur, fracture of the shaft of, ii. 224
 osteomyelitis of the upper shaft of,
 ii. 219
 Fever, hay, ii. 74
 malarial, iii. 69
 rheumatic, bacteriology of, i. 232
 typhoid, iii. 16
 hæmorrhagic pancreatitis in, i.
 271
 inoculation in, i. 270
 perforation of the intestine in,
 i. 270
 treatment of, iii. 1
 yellow, i. 228
 Filariasis, the blood in, iv. 258
 Finger, Ernest, treatment of acute ure-
 thritis, ii. 28
 Fire-arms at short range, effects of, iii.
 148
 Fixation of the right kidney, ii. 238
 Flail-joint, apparatus for, ii. 221
 Flaxseed tea for colds, iv. 40
 Flea, the house, iii. 119
 the sand- or jigger-, iii. 116
 Fleming, Robert A., experience gained
 from post-mortems in practice of medi-
 cine and surgery, ii. 142
 Folliculosis, iii. 229
 Food, passage of, iii. 256
 Formin, i. 254
 Fourth disease, the, i. 231; iii. 63
 Fracture, treatment of, ii. 42
 Fractures and dislocations and suits for
 malpractice, iii. 153
 Frontal sinus, opening of the, iv. 177
 Fusel-oil, poisoning by, i. 249

G

Gadfly, the human, iii. 110
 Gall-stones, i. 272
 hæmorrhage after operation for, i. 145
 Gardiner, Charles Fox, sanatory tent and
 its use in the treatment of pulmonary
 tuberculosis, iv. 1
 Gastric catarrh, iv. 7
 ulcer, hæmatemesis from, iv. 144
 prophylaxis of, i. 65
 surgical treatment of, i. 263

Gastro-enterostomy, *iii*. 180
 Gastro-intestinal auto-intoxication, *i*. 107 ;
 ii. 155
 tract, the blood in diseases of, *iv*. 267
 Gelatin for controlling hemorrhage, *i*. 251
 treatment of aneurisms by hypodermic
 injections of, *iv*. 36
 Genital organs in women, the blood in diseases
 of the, *iv*. 273
 Gersuny's method of prosthesis by subcutaneous
 and submucous injections of vaseline, *ii*. 8
 Gestation, extra-uterine, repeated, *i*. 276
 Gibbs, Charles, perforating bullet wounds
 of the central nervous system, *ii*. 203
 Glands, hæmolymp, importance of the,
 i. 283
 tuberculous, of the neck, *iv*. 166
 Glonoin, *i*. 250
 Glossolabial hemispasm, *iv*. 117
 Goitre, extirpation of, *i*. 265
 Gonorrhœa, *i*. 250
 the blood in, *iv*. 254
 Gonorrhœal myositis, *i*. 250
 Gout, the blood in, *iv*. 266
 Granule cells, *i*. 94
 Granules, basophilic, in red corpuscles, *i*.
 69
 origin of, *i*. 89
 Greig, David M., ectopia testis, *i*. 131
 Griffith, Frederic, a case of localized hypertrichosis;
 traumatism of the elbow
 stimulating fracture, followed by infection;
 care necessary in handling syphilis, *i*. 175
 Growths, the nature of new, *iv*. 77
 Guacamphol, *i*. 254
 Gualakinol, *i*. 254
 Gunshot wound of the neck, *ii*. 221
 Gyromele, Turck's, and the X-rays in diagnosis
 of diseases of the stomach, *i*. 240

H

Hæmatemesis, surgical treatment of, *iv*.
 144
 Hæmatoma, resection of a corpus-luteum,
 ii. 240
 Hæmoglobin, estimation of, *iv*. 214
 Hæmolymp glands, *i*. 283
 Hæmoptysis, parasitic, *i*. 289
 Hall, J. N., scars and marks of clinical
 interest, *iv*. 71
 Hallopeau, H., the treatment of acne, *i*. 54
 the urticarias; their causes, varieties,
 and treatment, *iii*. 45
 Hay fever, *ii*. 74
 Headache powders, *iv*. 43
 Heart and lungs from a case of left-sided
 pneumonia, *iv*. 51
 diseases of the, suprarenal capsule in,
 i. 250
 massage in narcosis, *iv*. 174

Hedonal, *i*. 254
 Helthin in testing purity of drinking
 water, *i*. 284
 Hemiplegia, functional and organic, *iv*.
 112
 syphilitic, *iv*. 122
 Hemmeter, John C., gastro-intestinal auto-
 intoxication, *i*. 107 ; *ii*. 155
 Hemorrhage after operation for gall-
 stones, *i*. 145
 cerebral, *iii*. 134
 gelatin for controlling, *i*. 251
 Hemorrhagic pancreatitis in typhoid fever,
 i. 271
 Hemorrhoids, *iii*. 175
 Henry, Frederick P., lungs and heart from
 a case of left-sided pneumonia with purulent
 pericardial effusion, *iv*. 51
 Hepatic sclerosis, *ii*. 102
 Hernia, radical cure of, *ii*. 189
 sliding, *ii*. 199
 suture material in, *iv*. 140
 Hinsdale, Guy, biographical sketches of
 eminent living physicians, *i*. 1 ;
 ii. 247 ; *iv*. 198
 climate of New England, *i*. 49
 Hints, practical, *i*. 64 ; *iii*. 63
 Holmes, Bayard, a primary evidment of
 tuberculous bone foci at the knee; sudden
 death after enucleation of thyroid
 gland for exophthalmic goitre; suppression
 of urine and hemorrhage after operation
 for gall-stones; a calculus in the
 appendix mistaken for ureteral calculus,
 i. 135
 Hopkins, S. D., bilateral cerebral thrombosis
 due to atheromatous changes in the
 arteries, *iii*. 123
 Hospital, modern private, *ii*. 261
 Hot air for cauterization, *iii*. 65
 House-flea, *iii*. 119
 Humerus, fracture of the external condyle
 of, *ii*. 224
 Hydrocele of the canal of Nuck, *ii*. 199
 Hypertrichosis, localized, *i*. 175
 Hysterical mutism, *iv*. 133

I

Ichthalbin, *i*. 255
 Ichthargon, *i*. 255
 Immunity, *iv*. 275
 Infantile palsy, surgical treatment of, *i*.
 180
 paralysis, *i*. 182
 followed by talipes calcaneus, *i*.
 184
 followed by talipes equino-valgus,
 i. 185
 spinal paralysis, *iii*. 142
 Infections with colon bacilli, serum diagnosis
 of, serum therapy in, and immunity from,
 iv. 297
 Infectious diseases, the blood in, *iv*. 248

Inflammation, *iii*. 268
 Inflammatory processes, the blood in, *iv*. 260
 Influenza, the blood in, *iv*. 254
 Infusion in general practice, *i*. 67
 Inguinal hernia, radical cure of, *iv*. 136
 region, anatomy of the, *iv*. 136
 Inoculation in typhoid fever, *i*. 270
 Insane witnesses, *i*. 245
 Insanity, *i*. 244
 and nervous diseases, *i*. 245
 Insect pests of human beings, *iii*. 107
 Instruments, new, *i*. 294
 Intestinal juice, the, *iii*. 262
 parasites, diseases due to, the blood in, *iv*. 258
 perforation in typhoid fever, *i*. 268; *iii*. 16
 Intestine, perforation of the, in typhoid fever, *i*. 270; *iii*. 16
 resection of the, *iv*. 178
 Intoxications, the blood in, *iv*. 272
 Iodocrol, *i*. 255
 Iodomuth, *i*. 255
 Ipecacuanha spray for winter cough, *iii*. 66
 Itrol, *i*. 255

J

Jardine, Robert, the treatment of cases of face presentation, *iii*. 213
 Jaundice, the blood in, *iv*. 274
 Jigger-flea, the, *iii*. 116
 Jonnesco, Thomas, resection of the cervical sympathetic, *ii*. 177

K

Keen, William W., biographical sketch of, *iv*. 202
 Kelly, Aloysius O. J., some clinical aspects of aneurisms of the aorta, *iv*. 81
 Kelly, H. A., nephrectomy; panhysteromyomectomy, with double salpingo-oophorectomy and appendicectomy; fixation of right kidney; suspension of uterus, with resection of a corpus luteum hæmatoma; salpingo-oophorectomy, followed by resection of the sigmoid for carcinoma, *ii*. 233
 some notes upon the management of a modern private hospital, *ii*. 261
 Kelly, James, and James K. Young, the surgical treatment of infantile palsy, *i*. 180
 Kidney, decapsulation of the, *i*. 262
 fixation of the right, *ii*. 238
 Koenig, Charles J., treatment of deafness by direct massage of the ossicles of the ear, *iii*. 56

L

Labor, the blood in, *iv*. 271
 Lancereaux, E., treatment of aneurisms by gelatin in hypodermic injections, *iv*. 36
 Largin, *i*. 255
 Larynx, malignant diseases of, *iv*. 28
 Lead palsy and trauma, *iv*. 126
 poisoning, *i*. 87
 the blood in, *iv*. 272
 Legeu, F., two cases of immediate death caused by the spinal injection of cocaine, *ii*. 67
 Legislation, medical, *i*. 242
 Lemonade, nutritive, *iv*. 45
 Lépine, R., on the treatment of diabetes mellitus, *ii*. 1
 Leprosy, serum diagnosis of, serum therapy in, and immunity from, *iv*. 296
 Leser, Edmund, artificial respiration and heart massage in narcosis; Volkmann's suspension apparatus; ligature of the common carotid artery; opening of the frontal sinus; resection of the superior and inferior orbital nerves; suturing of the bowel, *iv*. 174
 Lesions, traumatic, of the brain, *iv*. 102
 Leucocythæmia, splenomyelogenic, subsequent to an attack of malarial fever, *iii*. 69
 Leucocytosis, *iv*. 243
 neutrophilic, *iv*. 245
 polymorphonuclear eosinophilic, *iv*. 246
 Leucopenia, *iv*. 247
 Leukæmia, *iii*. 69; *iv*. 236
 Lewis, John A., the treatment of fractures and dislocations in relation to suits for malpractice, *iii*. 153
 Ligature of the common carotid artery, *iv*. 175
 of the posterior tibial artery, *iv*. 176
 Liquid air, *i*. 252
 Liver, cirrhosis of, *i*. 266; *ii*. 92
 protective function of, *i*. 122
 Lucas-Champlonnière, passive movements and massage for the treatment of fracture, *ii*. 42
 Lumbar puncture, new methods of diagnosis, *i*. 261
 Lungs and heart from a case of left-sided pneumonia, *iv*. 51
 tuberculosis of the, *iv*. 56
 Lymphocytosis, *iv*. 247
 Lysoform, *i*. 255

M

Malaria, *i*. 220
 and cancer, *i*. 223
 destruction of mosquitoes, *i*. 222
 remedies for, *i*. 222
 the blood in, *iv*. 257

- Malarial fever, followed by splenomyelogenous leucocythæmia, *iii.* 69
nephritis, *i.* 223
Malpractice, *iii.* 153
Malta fever, serum diagnosis of, serum therapy in, and immunity from, *iv.* 296
the blood in, *iv.* 254
Mania, acute, following injury, *iii.* 131
Marks and scars, *iv.* 71
Marnoch, John, coxa vara, *i.* 127
Massage and passive movements in the treatment of fracture, *ii.* 42
for deafness, *iii.* 56
heart, in narcosis, *iv.* 174
Mastoid disease following smallpox, *i.* 193
Mastoiditis, acute, *iii.* 233
Mauclaire, P., the diagnosis and treatment of osteomyelitis, *iii.* 86
Mauger, Noel, treatment of intestinal perforation in typhoid, *iii.* 16
McKinley, President, death of, *i.* 300
McPhedran, Alexander, dilatation of the stomach, *i.* 96
Measles, the blood in, *iv.* 252
Medical Department, United States Army, *ii.* 249
legislation, *i.* 242
subjects, general, *i.* 226
Medicine, *i.* 201; *ii.* 81; *iii.* 69; *iv.* 47
progress of, during the year 1901, *i.* 198
Meigs, Arthur V., use of opium in daily practice, *i.* 7
Meloplasty, *iv.* 164
Membrane, diphtheritic, histological characteristics of, *i.* 286
Meninges, permeability of the, *i.* 261
Menstruation, first, *i.* 275
Mental preoccupation as a preliminary to general anæsthesia, *iv.* 154
Mercuroi, *i.* 255
Milk supply, child mortality and, in Chicago, *i.* 229
Mitchell, S. Weir, biographical sketch of, *i.* 1
Morphine poisoning, adrenalin in, *i.* 232
Morphinism, treatment of, *iii.* 24
Mosquitoes, destruction of, *i.* 222
Motor anomalies of the eye, table of, *iv.* 191
Mouth-wash, antiseptic, *ii.* 79
Moynihan, Berkeley G. A., the surgical treatment of hæmatemesis from gastric ulcer, *iv.* 144
Müller, L. R., treatment of bladder and rectal troubles in nervous diseases, *ii.* 15
Munson, E. L., an outline of the organization and work of the medical department of the United States Army, *ii.* 249
Murphy, John B., biographical sketch of, *ii.* 247
Mutism, hysterical, *iv.* 133
Myasthenia gravis and polyorrhomenitis, *i.* 67
- N**
- Nabarro, David, a case of splenomyelogenous leucocythæmia subsequent to an attack of malarial fever, *iii.* 69
Narcosis, artificial respiration and heart massage in, *iv.* 174
chloroform, fright and deaths in, *iv.* 153
Nargol, *i.* 255
Nasal discharge, *ii.* 78
Neck, tuberculous glands of the, *iv.* 166
Nephrectomy, *ii.* 233
Nephritis, malarial, *i.* 223
the blood in, *iv.* 264
Nerves, superior and inferior, resection of the, *iv.* 178
Nervous diseases, *ii.* 15
and insanity, *i.* 245
system, bullet wounds of, *ii.* 203
effects of drugs upon the, *i.* 41
Neuritis, arsenical, *i.* 233
Neurology, *i.* 244; *iv.* 97
Neuronic architecture of the visual apparatus, *i.* 247
Neuroses, the functional, the blood in, *iv.* 269
New England, climate of, *i.* 49
New instruments and devices, *i.* 293
remedies, *i.* 252
Nirvanin, *i.* 255
Nitrous oxide and ether, *i.* 258
Nursing-bottle, nipple of the, *i.* 231
- O**
- Obstetrics, *i.* 187; *ii.* 233; *iii.* 206
Obstipation, *iii.* 173
Oophorectomy and thyroid medication, *i.* 278
Opening of the frontal sinus, *iv.* 177
Opium poisoning, adrenalin in, *i.* 232
use of, in daily practice, *i.* 7
Orbital nerves, superior and inferior, resection of the, *iv.* 178
Osborne, O. T., treatment of typhoid fever, *iii.* 1
Osteomyelitis, diagnosis and treatment of, *iii.* 86
of the upper shaft of the femur, *ii.* 219
Ovarian grafting, *i.* 274
Ozæna, citric acid for, *i.* 64
- P**
- Pachymeningitis hæmorrhagica, *ii.* 81
Packard, Francis R., the faucal tonsils; the indications for their removal and the best methods by which to accomplish it, *iii.* 240

- Packard, the late Frederick A., pernicious anemia with extensive pigmentary changes in the skin, iv. 47
- Palate, cleft, and harelip, i. 155
- Palsy, infantile, surgical treatment of, i. 180
- lead, and trauma, iv. 126
- Pancreas, function of the, iii. 247
- Pancreatic cysts, ii. 113
- Pancreatitis, acute, i. 226
- hemorrhagic, in typhoid fever, i. 271
- Panhystero-myomectomy, ii. 236
- Pantograph, chest, i. 294
- Paralysis, i. 180; iv. 126, 181
- general, the blood in, iv. 269
- infantile spinal, iii. 142
- Paranoia, iv. 97
- Parasites, diseases due to, the blood in, iv. 258
- Parasitic hæmoptysis, i. 289
- Patella, fracture of, ii. 222
- Pathology, comparative, i. 287
- Patton, Joseph M., bradycardia; cyclic albuminuria, ii. 148
- Pavlov, I. P., function of the digestive glands, ii. 274; iii. 247
- Pennington, J. Rawson, obstipation; hemorrhoids; fissure ani, iii. 173
- Percussion, auscultatory, ii. 102
- renal, iii. 66
- trimanual, i. 276
- Perforation, intestinal, in typhoid fever, i. 268
- Peritonitis, tuberculous, i. 64
- Pertussis, the blood in, iv. 249
- serum diagnosis of, serum therapy in, and immunity from, iv. 298
- Phlyctenular eruption, recurring, of the fingers, iv. 180
- Pick, Arnold, pachymeningitis hæmorrhagica as a cause of drunkards' deaths, ii. 81
- Piles, internal, iii. 163
- Pill, tonic, iv. 44
- Plague, i. 217
- the blood in, iv. 254
- Plastic operation on the right foot, ii. 222
- Pleurisy, i. 102
- serofibrinous, iii. 87
- Pneumlin, i. 255
- Pneumonia and pneumococcus infections, serum diagnosis of, serum therapy in, and immunity from, iv. 292
- left-sided, iv. 51
- the blood in, iv. 253
- water in, iii. 63
- Poisoning by aniline oil, i. 249
- by coal-tar naphtha, i. 249
- preparations, the blood in, iv. 272
- by fusel-oil, i. 249
- by lead, i. 87
- the blood in, iv. 272
- Poisoning by morphine and opium, adrenalin in, i. 232
- by rhus, ii. 73
- by sulfonal and trional, i. 249
- cobra, treated with Calmette's antivenine, i. 249
- thyroid, i. 83
- Polyorrhomenitis, myasthenia gravis and, i. 67
- Post-mortem experience, ii. 142
- Powders, headache, iv. 43
- Practical hints, i. 64; iii. 63
- Pregnancy, the blood in, iv. 271
- Prescription, simplified, writing and dosage for children, i. 251
- Prescriptions, selected, ii. 73; iv. 39
- Prolapse of the uterus and vagina, i. 274
- Prophylaxis of gastric ulcer, i. 65
- Prostatic abscess, iii. 68
- inflammation and hypertrophy, iii. 68
- Prothesis by injections of vaseline, ii. 8
- Pruritus, universal, ii. 80
- Pseudoleukæmia, ii. 89; iv. 241
- Puerperium, the blood in, iv. 271
- Pulmoform, i. 255
- Purpura hæmorrhagica, treatment of, i. 228
- the blood in, iv. 267
- Pyocyanous and proteus bacilli, serum diagnosis of, serum therapy in, and immunity from, iv. 297

R

- Rabies, rapid diagnosis of, i. 291
- Randall, B. Alex., deposit of chalk in the tympanic membrane; a case of mastoid disease following smallpox, i. 192
- Rectal and bladder troubles, ii. 15
- disease, i. 169
- Remedies, new, i. 252
- Renal disease, skiagraphic diagnosis in, i. 242
- percussion, iii. 66
- Resection of intestine, iv. 178
- of the superior and inferior orbital nerves, iv. 178
- of tuberculous testicle, iv. 161
- Respiration, artificial, in narcosis, iv. 174
- Rheumatic discomfort in the old, iv. 44
- fever, bacteriology of, i. 232
- Rheumatism, ii. 76
- acute articular, i. 228
- chronic, iv. 42
- the blood in, iv. 256
- Rhus poisoning, ii. 73
- Rinderpest, i. 293
- Robin, Albert, treatment of simple ulcer of the stomach, ii. 54
- Rodman, William L., removal of superior maxilla; operation for cleft palate and harelip; tetanus; tubercular disease of right wrist; operation for laceration

- of internal and external sphincter muscles; rectal disease; removal of a dermoid cyst; round-celled sarcoma; anal fistula, i. 151
- Romme, R., Gersuny's method of prosthesis by subcutaneous and submucous injections of vaseline, ii. 8
- S**
- Salpingo-oophorectomy, ii. 236
- Sand-flea, the, iii. 116
- Santi, Philip R. W. de, some practical points on the early diagnosis and treatment of malignant disease of the larynx, iv. 28
- Sarcoma, mixed toxins for, ii. 201
round-celled, i. 171
- Scarlet fever, the blood in, iv. 252
- Scars and marks, iv. 71
- Schlapp, Max, syphilitic hemiplegia; lead palsy and trauma; anomalous general paralysis; hysterical mutism, iv. 122
- Schmaus, Hans, a critical study of the theory of inflammation, iii. 268
- Schneiderlin, method of, i. 259
- Sclerosis, hepatic, ii. 102
- Scurvy, infantile, i. 229
- Sedative, uterine, iv. 43
- Selected prescriptions, ii. 73
- Senn, N., osteomyelitis of the upper shaft of the femur; appendicitis with ulcerative cæcitis; gunshot wound of the neck; tuberculous arthritis of the elbow-joint; plastic operation on the right foot; anterior arthrotomy; fracture of the patella; fracture of the external condyle of the humerus; fracture of the shaft of the femur; vesical calculus, iii. 219
- Serofibrinous pleurisy, iii. 87
- Serum diagnosis, iv. 275; iv. 297
reaction, iv. 216
therapy, iv. 275
and immunity, iv. 298
- Shoemaker, William T., the clinical significance of binocular diplopia, iv. 188
- Sigmoid, resection of, for carcinoma, ii. 243
- Silver wire and electricity in the treatment of aortic aneurism, i. 236
- Simon, Charles E., on the significance of basophilic granules in red corpuscles, with special reference to their recurrence in chronic lead poisoning, i. 69
- Sinus, frontal, opening of the, iv. 177
- Skiagraphic diagnosis in renal and ureteral disease, i. 242
- Skin, the blood in diseases of the, iv. 271
- Skin-stretching, i. 232
- Smallpox, i. 211
followed by mastoid disease, i. 193
the blood in, iv. 255
- Sphincter muscles, operation after laceration of, i. 164
- Spiller, William G., traumatic lesions of the brain in their relation to operation, iv. 102
- Spinal anæsthesia, i. 259
injection of cocaine, ii. 67
paralysis, infantile, iii. 142
- Splenomegaly, primary, i. 271
- Splenomyelogenic leucocythæmia, iii. 69
- Stern, Heinrich, treatment of chronic gastric catarrh, iv. 7
- Stethophonometer, i. 293
- Stewart, Purves, the diagnosis of functional and organic hemiplegia, iv. 112
- Stieren, Edward, syphilis of the eye, iii. 218
- Stomach, dilatation of, i. 96; iii. 180, 187
surgical intervention in, iii. 187
diseases of, i. 240
simple ulcer of, ii. 54
- Streptococcus, serum diagnosis of, serum therapy in, and immunity from, iv. 297
- Stricture, ii. 75
- Stye, abortive treatment of, iii. 67
- Sulfonal, poisoning by, i. 249
- Superior maxilla, removal of, i. 151
- Suprarenal capsule in diseases of the heart, i. 250
- Surgery, i. 127; ii. 178; iii. 148; iv. 136
- Surgical intervention in dilatation of the stomach, iii. 187
subjects, i. 262
treatment of hæmatemesis, iv. 144
- Suspension apparatus, Volkmann's, iv. 175
- Suture material in hernia operations, iv. 140
- Sweating of the feet, iv. 45
- Sympathetic, cervical, resection of, ii. 177
- Symphiseotomy and Cæsarean section, i. 187
- Syphilis of the eye, iii. 218
the blood in, iv. 252
- Syphilitic hemiplegia, iv. 122
- Syphilitics, i. 177
- Syringomyelia, iii. 131
- T**
- Tannalbin, i. 255
- Tannigen, i. 255
- Tannoform, i. 255
- Tapeworm, a new, i. 283
- Taylor, P. Richard, folliculosis; trachoma; removal of cataract, iii. 229
- Tent, sanatory, for treatment of pulmonary tuberculosis, iv. 1
- Testicle, tuberculous, resection of, iv. 161
- Tetanus, i. 158
serum diagnosis of, serum therapy in, and immunity from, iv. 291
treatment of, i. 160, 235; iv. 291

- Thayer, Addison S., to what extent can the general practitioner make use of the newer diagnostic methods? *iii.* 81
- Therapeutics, *i.* 7, 250; *ii.* 1; *iii.* 1; *iv.* 1
- Thrombosis, bilateral cerebral, *iii.* 123
- Thyroid gland, enucleation of, for exophthalmic goitre, *i.* 141
relation of, to autointoxication, *ii.* 162
poisoning, *i.* 33
- Thyroidectomy, the blood in, *iv.* 274
- Tibial artery, posterior, ligature of the, *iv.* 176
- Tonsillitis, the blood in, *iv.* 256
- Tonsils, the faucial, *iii.* 240
- Toxicology, *i.* 248
- Toxins, mixed, for inoperable sarcoma, *ii.* 201
- Trachoma, *iii.* 229
- Trauma and lead palsy, *iv.* 126
- Traumatic lesions of the brain, *iv.* 102
- Traumatism of the elbow, *i.* 176
- Treatment, abortive, of sty, *iii.* 67
of aneurisms by gelatin in hypodermic injections, *iv.* 36
of anomalous general paralysis, *iv.* 133
of chronic gastric catarrh, *iv.* 7
of deafness, *iii.* 56
of hysterical mutism, *iv.* 135
of intestinal perforation in typhoid, *iii.* 16
of lead palsy, *iv.* 129
of malignant disease of the larynx, *iv.* 28
of morphinism, *iii.* 24
of osteomyelitis, *iii.* 24
of pulmonary tuberculosis in sanatory tent, *iv.* 1
of syphilitic hemiplegia, *iv.* 125
of the urticarias, *iii.* 45
of typhoid fever, *iii.* 1
surgical, of hæmatemesis, *iv.* 144
- Trichinosis, the blood in, *iv.* 258
- Trional, poisoning by, *i.* 249
- Tuberculosis, *i.* 201
diseases influencing, *i.* 210
legislation controlling, recent, *i.* 209
of the lungs and diseases which resemble it, *iv.* 56
persistent diarrhœa in, *ii.* 73
pulmonary, the sanatory tent in, *iv.* 1
serum therapy in, *iv.* 288
the blood in, *iv.* 248
treatment of, with urea, *i.* 210
tuberculin in diagnosis and treatment of, *i.* 210
urea in, *i.* 250
- Tuberculous arthritis of the elbow-joint, *ii.* 221
bone foci at the knee, *i.* 135
disease of right wrist, *i.* 162
persistent diarrhœa in, *ii.* 73
- Tuberculous fistulæ, *iii.* 168
glands of the neck, *iv.* 166
peritonitis, *i.* 64
testicle, resection of, *iv.* 161
- Tumor, abdominal, *iii.* 206
- Turck's gyromele and the X-rays in diagnosis of diseases of the stomach, *i.* 240
- Tuttle, James F., internal piles; the clamp and cautery operation; tuberculous fistulæ, *iii.* 163
- Tympanic membrane, deposit of chalk in, *i.* 192
- Typhoid fever, *iii.* 16
diagnosis of, *iv.* 279
hemorrhagic pancreatitis in, *i.* 271
inoculation in, *i.* 270
perforation of the intestine in, *i.* 270
the blood in, *iv.* 252
treatment of, *iii.* 1
by serum therapy, *iv.* 279
- U**
- Ulcer, gastric, hæmatemesis from, *iv.* 144
prophylaxis in, *i.* 65; *ii.* 54
simple, treatment of, *i.* 54
surgical treatment of, *i.* 263
- Ulcers, varicose, prophylaxis of, *ii.* 80
treatment of, *ii.* 80
- Uncinariosis, the blood in, *iv.* 259
- United States Army, medical department of, *ii.* 249
- Urea in tuberculosis, *i.* 250
- Ureteral disease, skiagraphic diagnosis in, *i.* 242
- Urethritis, acute, *ii.* 28, 35, 39
- Urine, green or blue, *i.* 284
suppression of, *i.* 145
- Urosine, *i.* 256
- Urticarias, the, *iii.* 45
- Uterine atresia, *i.* 275
sedative, *iv.* 43
- Uterus, prolapse of the, *i.* 274
suspension of, *ii.* 240
- V**
- Vaccination, *iv.* 279
- Vagina, prolapse of the, *i.* 274
- Van Harlingen, Arthur, recurring phlyctenular eruption of the fingers, with changes in the nails, possibly of hysterical origin, *iv.* 180
- Varicose ulcers, prophylaxis of, *ii.* 80
treatment of, *ii.* 80
- Vaseline, prosthesis by injections of, *ii.* 8
- Veldt sore, *i.* 293
- Vesical calculus, *ii.* 226
- Vickery, Herman F., pseudoleukæmia with chronic relapsing fever, *ii.* 89
- Vioform, *i.* 256
- Virchow, Rudolph, *i.* 299

Visual apparatus, neuronie architecture of the, i. 247
 Volkmann's suspension apparatus, iv. 175

W

Walker, John B., treatment of acute appendicitis, ii. 213
 Walsh, James J., insect pests of human beings, iii. 107
 Ward, J. M., thyroid poisoning, i. 33
 Water in pneumonia, iii. 63
 use of helthm in testing, i. 284
 Watson, Edward Willard, progress of medicine during the year 1901, i. 198
 Whooping-cough, i. 65; iv. 44
 Willard, De Forest, club-foot; astragalectomy for relapsed and inveterate cases, iii. 201
 Winter cough, ipecacuanha spray for, iii. 66
 Witnesses, insane, i. 245
 Wood alcohol, blindness from, i. 248

Wood, Horatio C., biographical sketch of, iv. 198

Wood, Horatio C., a description of the methods of investigating the action of drugs, i. 37

Wyeth, John A., biographical sketch of, i. 4

X

X-rays in diagnosis of diseases of the stomach, i. 240
 in medicine, i. 241
 present status of, i. 239

Y

Yellow fever, i. 223
 organism of, i. 226
 the blood in, iv. 254

Yohimbe, i. 256

Young, James K., and James Kelly, the surgical treatment of infantile palsy, i. 180

END OF VOLUME IV.

THE ANÆMIAS

yield readily to organic, or true animal iron treatment.

A resort to *inorganic* iron preparations or tonics, serves only to stimulate corpuscular proliferation without supplying sufficient nutrition to mature the blood cells.

A preparation of **TRUE ANIMAL IRON** that will supply every deficiency in the blood, and assure the proliferation of *all* the corpuscles to a full and sturdy maturity, is found in

BOVININE

It contains **10% ANIMAL IRON**, 20% coagulable albumen, and every element of nutrition of the animal, mineral, and vegetable kingdoms.

It is readily absorbed by the tissues, requires little or no digestion, is prompt and reliable in stimulation and support, and is a nutrient of the very highest value.

BOVININE administration causes quick increase of the leucocytes, and a consequent arrest of all pathological processes.

BOVININE is advertised to the Profession only, and is a strictly ethical physician's preparation. Its formula is open to all.

A postal request brings you our Hand-book on Haematherapy, giving valuable information to both the general practitioner and the specialist.

THE BOVININE COMPANY,
75 W. HOUSTON ST., NEW YORK.

The Practice of Surgery

BY

HENRY R. WHARTON, M.D.,

University of Pennsylvania,

AND

B. FARQUHAR CURTIS, M.D.,

University and Bellevue Medical College, N. Y.

Third Edition. 1244 pages. Magnificently illustrated with full-page plates in colors and 924 text illustrations. 8vo.

Cloth, \$6.50; sheep, \$7.50

Delivered.

MANY paragraphs contain more information than pages of other works to which the profession is accustomed; the cuts and illustrations are very numerous and highly instructive, the chapters on amputation, fractures, and bandaging being so fully illustrated that the idea of the authors is at once apparent. Great pains have been taken in condensing a large amount of information, and in this manner space is found to include all the subjects which should find a place in a surgical treatise; even abdominal operations and others that are rarely performed except by the most experienced surgeons are lucidly described.

The relative proportions assigned to the various topics have been so carefully considered that the work is not top-heavy in any one direction, as is so often the case with many text-books, but it is one of the most eminently practical, complete, and best books for the general practitioner published.

Genito-Urinary and Venereal Diseases

BY

J. WILLIAM WHITE, M.D.,

Professor of Clinical Surgery, University of Pennsylvania,

AND

EDWARD MARTIN, M.D.,

Clinical Professor of Genito-Urinary Diseases, University of Pennsylvania.

Fifth Edition. Illustrated with 248 engravings and 7 colored plates. 8vo. 1061 pages.

Cloth, \$6.00; sheep, \$7.00

Delivered.

THAT five editions have been required in four years amply demonstrates the value of this standard work.

The most concise, lucid, thorough, modern, and practical book on the subject in the English language.

Those portions which deal with symptomatology and diagnosis are unusually full, and the illustrations are more numerous than is usual in works on this subject (many being photographs from life).

In treatment, descriptions of manipulations and operations are given with such detail that those who have not had practical experience may be enabled to carry them out.

All the practical points embraced under the general heading of Psychopathia Sexualis are carefully given, while genito-urinary asepsis and antisepsis are so simplified and clearly stated that they are made practicable for every physician.

New Complete Medical Portrait Catalogue sent free to any address

Publishers

J. B. Lippincott Company

Philadelphia

**SUPPLEMENT
TO KEATING'S**

Cyclopaedia of the Diseases of Children

MEDICAL AND SURGICAL

Edited by

**WILLIAM A. EDWARDS,
M.D.**

The articles written especially for the work by American, British, and Canadian authors. Second edition, revised. This fifth volume contains 1350 pages, with illustrations. Royal 8vo.

Cloth, \$5.00; sheep, \$6.00

New York Medical News:

"It is a supplement to Keating's masterpiece, and is up to date in every respect. All in all the volume is a most welcome one. It is not only a supplement to 'Keating's Cyclopaedia' but to every book on children's diseases and internal medicine."

A PRACTICAL TREATISE ON

Smallpox

Illustrated by Colored Photographs from Life

**By GEORGE HENRY FOX,
A.M., M.D.,**

Consulting Dermatologist to the New York
City Department of Health.

WITH THE COLLABORATION OF

**S. DANA HUBBARD, M.D.,
SIGMUND POLLITZER, M.D.,
JOHN H. HUDDLESTON, M.D.**

10 colored plates, 9½ x 12¼ inches. 6
black and white plates. 43 illustrations in all.

**Two portfolios . . \$3.00
Flexible cloth . . \$4.00
Flexible leather . \$5.00**

Pediatrics

**THE HYGIENIC AND MEDICAL
TREATMENT OF CHILDREN**

BY

**THOMAS MORGAN ROTCH,
M.D.,**

Professor of the Diseases of Children,
Harvard University.

Third edition, rearranged and re-written. Illustrated by 528 photo-engravings and 9 colored plates, containing 62 subjects. 1050 pages. 8vo.

Cloth, \$6.00; sheep, \$7.00

Medical News, New York:

"For many years this treatise has been one of the best classics in the English language, treating with the development and care of children in sickness and in health. Dr. Rotch has not limited himself to the study of disease processes alone; he has given almost as much space to the hygienic care of the child during health as to the study of disease conditions."

Journal of American Medical Association, Chicago:

"The physician who makes himself master of 'Pediatrics' will have a knowledge that will reach the heart and assist him in obtaining the confidence of the most important member of the average family—the mother—and thus be the greatest aid in establishing him in the opinions of those who can make or mar his success. The work before us is one that it is a pleasure to recommend to those who desire a book to aid them in obtaining this knowledge. It is scientific, it is practical, and will be found as helpful to the general practitioner as to the student."

Pacific Medical Journal, San Francisco, Cal.:

"Professor Rotch passes in review a young human being from birth until adolescence; his feeding, clothing, growth, and the many diseases incident to his period of life. The work is a masterpiece and should be possessed by every student and practitioner."

New Complete Medical Portrait Catalogue sent free to any address

Publishers

J. B. Lippincott Company

Philadelphia

3 CAL 1 1/2
COUNTWAY LIBRARY
HC 2EKQ 5

